FEBRUARY 2000
VOLUME 45
NUMBER 2
ISSN 0020-1324-RECACP

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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
45TH YEAR—ESTABLISHED 1956

SPECIAL ISSUE

LONG-TERM OXYGEN THERAPY PART II

- LTOT in Conditions Other Than COPD
- LTOT vs Long-Term Ventilatory Assistance
- Hypoxemia During Sleep
- Exercise Hypoxemia
- Ambulatory Oxygen Therapy, Exercise, and Survival with Advanced COPD
- Oxygen and Air Travel
- Demographics and Economics
- A Nationwide System of LTOT: The Polish Experience
- LTOT Conference Summary

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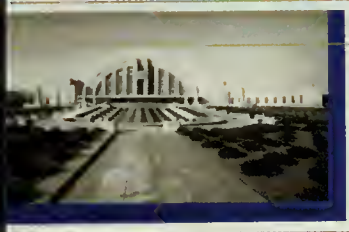

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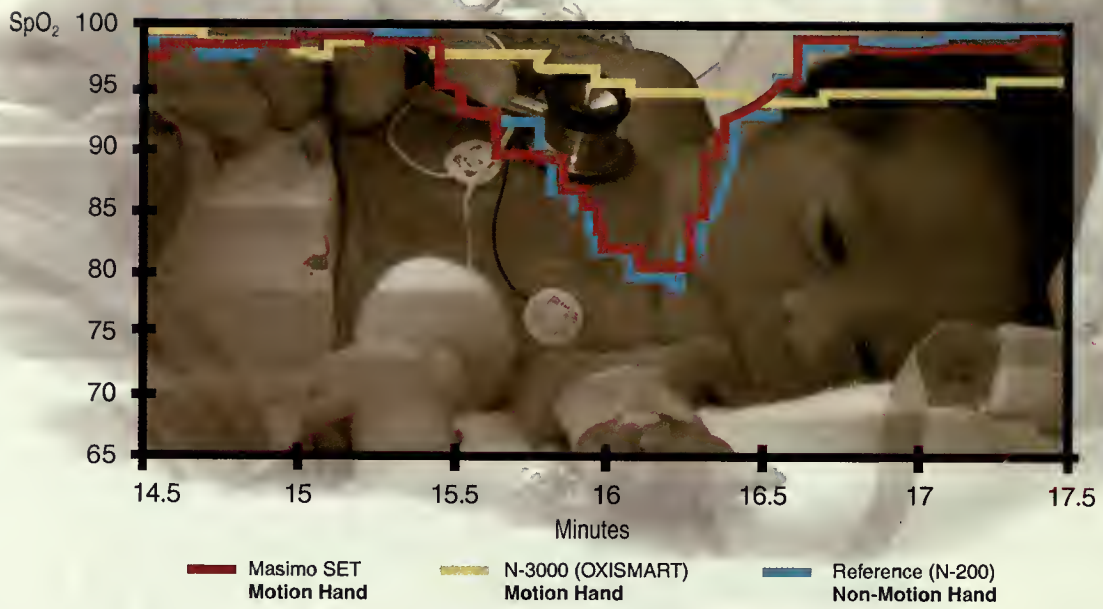

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low perfusion study

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RESPIRATORY CARE (ISSN 0020-1324, USPS 0489-190) is published monthly by Daedalus Enterprises Inc, at 11030 Ables Lane, Dallas TX 75229-4593, for the American Association for Respiratory Care. One volume is published per year beginning each January. Subscription rates are $75 per year in the US; $90 in all other countries (for airmail, add $94).

The contents of the Journal are indexed in Hospital and Health Administration Index, Cumulative Index to Nursing and Allied Health Literature, EMBASE/Exerpta Medica, and RNdex Library Edition. Abridged versions of RESPIRATORY CARE are also published in Italian, French, and Japanese, with permission from Daedalus Enterprises Inc.

Periodicals postage paid at Dallas TX and at additional mailing offices. POSTMASTER: Send address changes to RESPIRATORY CARE, Membership Office, Daedalus Enterprises Inc, 11030 Ables Lane, Dallas TX 75229-4593.

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Mechanical Ventilation with PEEP: A Unique Therapy for Failing Hearts—Peters J. Intensive Care Med 1999 Aug;25(8):778–780.

Can It Work? Does It Work? Is It Worth It? The Testing of Healthcare Interventions Is Evolving—Haynes B. BMJ 1999 Sep 11;319(7211):652–653.

Rationale and Design of the National Emphysema Treatment Trial (NETT): A Prospective Randomized Trial of Lung Volume Reduction Surgery (review). J Thorac Cardiovasc Surg 1999 Sep;118(3):518–528.

Inhaled Nitric Oxide for Preterm Infants: Still an Experimental Therapy—Saugstad OD. Lancet 1999 Sep 25;354(9184):1047–1048.

Inhaled Nitric Oxide: Basic Biology and Clinical Applications—Steudel W, Hurford WE, Zapol WM. Anesthesiology 1999 Oct;91(4):1090–1121.

Nasal Nitric Oxide in Man—Lundberg JO, Weitzberg E. Thorax 1999 Oct;54(10):947–952.

Inhaled Nitric Oxide in Acute Lung Injury and Acute Respiratory Distress Syndrome: Inability to Translate Physiologic Benefit to Clinical Outcome Benefit in Adult Clinical Trials—Dellinger RP. Intensive Care Med 1999 Sep;25(9):881–883.

Sleep on the Cheap: The Role of Overnight Oximetry in the Diagnosis of Sleep Apnoea Hypopnoea Syndrome—Bennett JA, Kinnear WJ. Thorax 1999 Nov;54(11):958–959.

Hyperbaric Oxygen in Carbon Monoxide Poisoning: Conflicting Evidence That It Works—Weaver LK. BMJ 1999 Oct 23;319(7217):1083–1084.

Nocturnal Oximetry for the Diagnosis of the Sleep Apnoea Hypopnoea Syndrome: A Method To Reduce the Number of Polysomnographies?—Chiner E, Signes-Costa J, Arriero JM, Marco J, Fuentes I, Sergado A. Thorax 1999 Nov;54(11):968-971.

BACKGROUND: Polysomnography (PSG) is currently the "gold standard" for the diagnosis of the sleep apnoea hypopnoea syndrome (SAHS). Nocturnal oximetry (NO) has been used with contradictory results. A prospective study was performed to determine the accuracy of NO as a diagnostic tool and to evaluate the reduction in the number of PSGs if the diagnosis of SAHS had been established by this method. METHODS: Two hundred and seventy five patients with a clinical suspicion of SAHS were admitted to undergo, in the same night, full PSG and NO. Desaturation was defined as a fall in the haemoglobin saturation level (S_{aO_2}) to lower than 4% from the baseline level and an oxygen desaturation index per hour (ODI) was obtained in each patient with three cut off points: ≥ 5 (ODI-5), ≥10 (ODI-10), and ≥15 (ODI-15). RESULTS: SAHS was diagnosed in 216 patients (194 men). After withdrawing patients with abnormal lung function (forced expiratory volume in one second (FEV_1) lower than 80% predicted), sensitivity (SE), specificity (SP), positive and negative predictive values (PPV and NPV) of NO were: ODI-5 (80%, 89%, 97%, 48%); ODI-10 (71%, 93%, 97%, 42%); ODI-15 (63%, 96%, 99%, 38%). The accuracy for each ODI was 0.81, 0.75, and 0.70, respectively. If NO had been considered as a diagnostic tool and PSG had been performed only in patients with a negative NO (false negative and true negative) and those with a positive NO and abnormal pulmonary function tests, 135/275 (ODI-5), 156/275 (ODI-10), and 170/275 (ODI-15) PSGs would have been performed, a reduction of 140, 119, and 105, respectively. CONCLUSION: Nocturnal oximetry in patients with suspected SAHS and normal spirometric values permits the institution of therapeutic measures in most patients.

An Empirical Comparison of the St George's Respiratory Questionnaire (SGRQ) and the Chronic Respiratory Disease Questionnaire (CRQ) in a Clinical Trial Setting—Molken MR, Roos B, Van Noord JA. Thorax 1999 Nov; 54(11):995-1003.

BACKGROUND: The Chronic Respiratory Questionnaire (CRQ) and the St George's Respiratory Questionnaire (SGRQ) are the two most widely used quality of life questionnaires in chronic obstructive pulmonary disease

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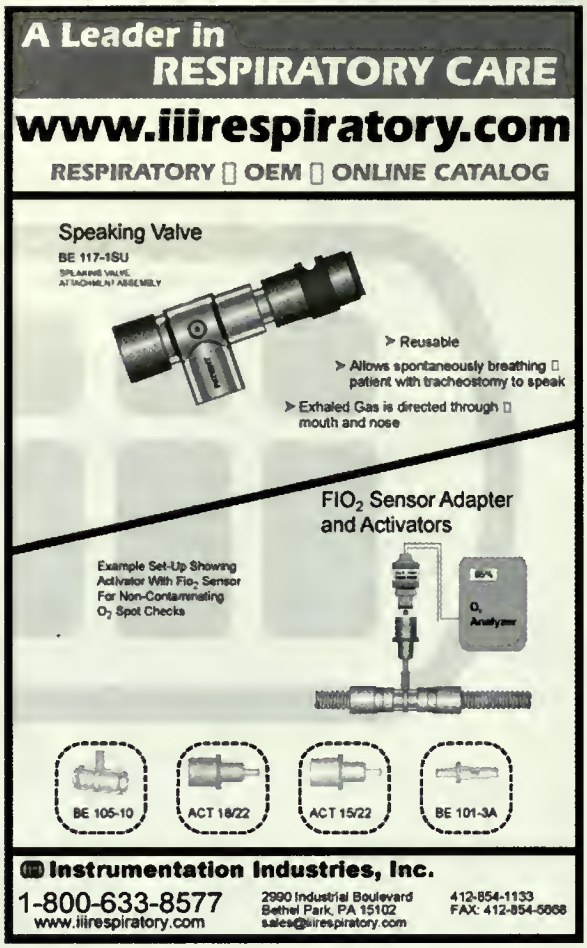

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(COPD). A study was undertaken to compare directly the self-administered version of the CRQ and the SGRQ with respect to feasibility, internal consistency, validity, and sensitivity to changes resulting from bronchodilator therapy. METHODS: One hundred and forty four patients with moderate or severe COPD were randomly assigned to receive three months of treatment with either salmeterol, salmeterol + ipratropium bromide, or placebo. Quality of life was measured at baseline and after 12 weeks of treatment. RESULTS: The proportions of missing values per patient were low for both questionnaires (0.54% for the CRQ and 2% for the SGRQ). The internal consistency was good for both questionnaires (Cronbach's alpha coefficients ≥0.84 for the CRQ and ≥0.76 for the SGRQ). Factor analysis confirmed the original domain structure of the CRQ but not of the SGRQ. Correlations with forced expiratory volume in one second (FEV_1) % predicted and peak expiratory flow rate (PEFR) were low for both questionnaires but better for the SGRQ than for the CRQ. The ability to discriminate between subjects with different levels of FEV_1 was somewhat better for the SGRQ. The correlations with symptom scores were comparable for both questionnaires. Cross sectionally, the scores of the two questionnaires were moderately to highly correlated (coefficients ranged from 0.35 to 0.72). Longitudinally, these correlations were lower (coefficients ranged from 0.17 to 0.54) but were still significant. The CRQ total and emotions score and the SGRQ symptoms score were the most responsive to change. The SGRQ symptoms domain was the only domain where the improvement in patients receiving combination treatment crossed the threshold for clinical relevance. CONCLUSIONS: Since this analysis of reliability, validity, and responsiveness to change did not clearly favour one instrument above the other, the choice between the CRQ and the SGRQ can be based on other considerations such as the required sample size or the availability of reference values.

Dietary Factors and Pulmonary Function: A Cross Sectional Study in Middle Aged Men from Three European Countries—Tabak C, Smit HA, Rasanen L, Fidanza F, Menotti A, Nissinen A, et al. Thorax 1999 Nov;54(11): 1021-1026.

BACKGROUND: Results of epidemiological studies relating individual dietary factors to chronic obstructive pulmonary disease (COPD) are inconsistent. To evaluate the cross sectional association of dietary factors with pulmonary function, data were collected from middle aged men in three European countries. METHODS: The data were collected in the 1960s in Finland (n = 1248), Italy (n = 1386), and the Netherlands (n = 691). Dietary intake was estimated using the cross-check dietary history method. Forced expiratory volume ($FEV_{0.75}$ or FEV_1, here called FEV) was measured by spirometry. Associations were adjusted for age, height, smoking, body mass index (BMI), alcohol consumption, and energy intake. RESULTS: FEV was positively associated with intake of vitamin E in Finland, with intake of fruit in Italy, and with intake of beta-carotene in the Netherlands. In all three countries men with intakes of both fruit and vegetables above the median had a higher FEV than those with a low intake of both foods. The difference in FEV ranged from 110 to 169 mL before and from 53 to 118 mL after energy adjustment. Differences in FEV for intake of three antioxidants (vitamins C and E and beta-carotene) above versus below the median ranged from 61 to 181 mL before and from -35 to 58 mL after energy adjustment. Intake of fish was not associated with FEV. CONCLUSIONS: In three European countries a high intake of fruit and vegetables was positively associated with pulmonary function. A high intake of all three antioxidants tended to be positively associated with pulmonary function before, but not after, adjustment for energy intake. Associations of individual antioxidants with pulmonary function were not consistent across countries.

A Randomized, Controlled Trial of the Effects of Remote, Intercessory Prayer on Outcomes in Patients Admitted to the Coronary Care Unit—Harris WS, Gowda M, Kolb JW, Strychacz CP, Vacek JL, Jones PG, et al. Arch Intern Med 1999 Oct 25;159(19):2273-2278.

CONTEXT: Intercessory prayer (praying for others) has been a common response to sickness for millennia, but it has received little scientific attention. The positive findings of a previous controlled trial of intercessory prayer have yet to be replicated. OBJECTIVE: To determine whether remote, intercessory prayer for hospitalized, cardiac patients will reduce overall adverse events and length of stay. DESIGN: Randomized, controlled, double-blind, prospective, parallel-group trial. SETTING: Private, university-associated hospital. PATIENTS: Nine hundred ninety consecutive patients who were newly admitted to the coronary care unit (CCU). INTERVENTION: At the time of admission, patients were randomized to receive remote, intercessory prayer (prayer group) or not (usual care group). The first names of patients in the prayer group were given to a team of outside intercessors who prayed for them daily for 4 weeks. Patients were unaware that they were being prayed for, and the intercessors did not know and never met the patients. MAIN OUTCOME MEASURES: The medical course from CCU admission to hospital discharge was summarized in a CCU course score derived from blinded, retrospective chart review. RESULTS:

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Compared with the usual care group (n = 524), the prayer group (n = 466) had lower mean ± SEM weighted (6.35 ± 0.26 vs 7.13 ± 0.27; p=0.04) and unweighted (2.7 ± 0.1 vs 3.0 ± 0.1; p=0.04) CCU course scores. Lengths of CCU and hospital stays were not different. CONCLUSIONS: Remote, intercessory prayer was associated with lower CCU course scores. This result suggests that prayer may be an effective adjunct to standard medical care.

Effects of Exercise Training on Older Patients with Major Depression—Blumenthal JA, Babyak MA, Moore KA, Craighead WE, Herman S, Khatri P, et al. Arch Intern Med 1999 Oct 25;159(19):2349-2356.

BACKGROUND: Previous observational and interventional studies have suggested that regular physical exercise may be associated with reduced symptoms of depression. However, the extent to which exercise training may reduce depressive symptoms in older patients with major depressive disorder (MDD) has not been systematically evaluated. OBJECTIVE: To assess the effectiveness of an aerobic exercise program compared with standard medication (ie, antidepressants) for treatment of MDD in older patients, we conducted a 16-week randomized controlled trial. METHODS: One hundred fifty-six men and women with MDD (age, ≥ 50 years) were assigned randomly to a program of aerobic exercise, antidepressants (sertraline hydrochloride), or combined exercise and medication. Subjects underwent comprehensive evaluations of depression, including the presence and severity of MDD using Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition criteria and Hamilton Rating Scale for Depression (HAM-D) and Beck Depression Inventory (BDI) scores before and after treatment. Secondary outcome measures included aerobic capacity, life satisfaction, self-esteem, anxiety, and dysfunctional cognitions. RESULTS: After 16 weeks of treatment, the groups did not differ statistically on HAM-D or BDI scores (p = 0.67); adjustment for baseline levels of depression yielded an essentially identical result. Growth curve models revealed that all groups exhibited statistically and clinically significant reductions on HAM-D and BDI scores. However, patients receiving medication alone exhibited the fastest initial response; among patients receiving combination therapy, those with less severe depressive symptoms initially showed a more rapid response than those with initially more severe depressive symptoms. CONCLUSIONS: An exercise training program may be considered an alternative to antidepressants for treatment of depression in older persons. Although antidepressants may facilitate a more rapid initial therapeutic response than exercise, after 16 weeks of treatment exercise was equally effective in reducing depression among patients with MDD.

Differences in Lung Bioavailability between Different Propellants for Fluticasone Propionate—Wilson AM, Sims EJ, Orr LC, Lipworth BJ. Lancet 1999 Oct 16;354(9187):1357-1358.

The lung bioavailability (as adrenal suppression) of fluticasone propionate was about twofold greater with chlorofluorocarbons than hydrofluoroalkane as propellant. Direct switching between formulations on a microg equivalent may therefore be inadvisable.

Impediments to Writing Do-Not-Resuscitate Orders—Eliasson AH, Parker JM, Shorr AF, Babb KA, Harris R, Aaronson BA, Diemer M. Arch Intern Med 1999 Oct 11;159(18):2213-2218.

BACKGROUND: Physicians are frequently unaware of their patients' desires regarding end-of-life care. Consequently, opportunities to implement do-not-resuscitate (DNR) orders are often missed. OBJECTIVE: To determine the reasons attending physicians do not write DNR orders when patients face increased mortality. METHODS: Over 4 months, the medical records

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of all inpatients on the General Medicine Service were reviewed at the time of discharge to identify patients with conditions predicting increased mortality. These cases were presented to a 5-member panel who decided if a DNR order was indicated. Reasons for missing DNR orders were discussed with the attending physicians. RESULTS: Of 613 consecutive admissions, the panel identified 149 patients (24%) for whom DNR orders were indicated. In 88 (59%) of these, DNR orders were absent. The lack of a DNR order did not correlate with age ($p = 0.95$), sex ($p = 0.61$), or race ($p = 0.80$). The attending physicians' explanations for not writing DNR orders in these 88 cases included the belief that the patient was not in imminent danger of death (n = 49 [56%]), the belief that the primary physician should discuss DNR issues (n = 43 [49%]), and the lack of an appropriate opportunity to discuss end-of-life issues (n = 38 [43%]). In 11 (12%) of the 88 cases, patients or their families did not accept the recommendation for a DNR order. No physicians expressed concerns regarding the morality of DNR orders, discomfort discussing end-of-life issues, or the threat of litigation as reasons for not writing a DNR order. CONCLUSIONS: Limitations in the extent and depth of the physician-patient relationship appear to be the most frequent impediments to writing DNR orders in our institution.

Are Health Care Professionals Advising Obese Patients to Lose Weight?—Galuska DA, Will JC, Serdula MK, Ford ES. JAMA 1999 Oct 27;282(16):1576-1578.

CONTEXT: Implementation of the National Institutes of Health's 1998 guidelines, which recommended that health care professionals advise obese patients to lose weight, required baseline data for evaluation. OBJECTIVES: To describe the proportion and characteristics of obese persons advised to lose weight by their health care professional during the previous 12 months and to determine whether the advice was associated with reported attempts to lose weight. DESIGN: The Behavioral Risk Factor Surveillance System, a random-digit telephone survey conducted in 1996 by state health departments. SETTING: Population-based sample from 50 states and the District of Columbia. PARTICIPANTS: A total of 12835 adults, 18 years and older, classified as obese (body mass index ≥ 30 kg/m^2), who had visited their physician for a routine checkup during the previous 12 months. MAIN OUTCOME MEASURES: Reported advice from a health care professional to lose weight, and reported attempts to lose weight. RESULTS: Forty-two percent of participants reported that their health care professional advised them to lose weight. Using multivariate logistic regression analysis, we found that the persons who were more likely to receive advice were female, middle aged, had higher levels of education, lived in the northeast, reported poorer perceived health, were more obese, and had diabetes mellitus. Persons who reported receiving advice to lose weight were significantly more likely to report trying to lose weight than those who did not (OR, 2.79; 95% CI, 2.53-3.08). CONCLUSIONS: Less than half of obese adults report being advised to lose weight by health care professionals. Barriers to counseling need to be identified and addressed.

Evaluation of the Effect of a Large Volume Spacer on the Systemic Bioactivity of Fluticasone Propionate Metered-Dose Inhaler—Dempsey OJ, Wilson AM, Coutie WJ, Lipworth BJ. Chest 1999 Oct;116(4):935-940.

Background: Inhaled corticosteroids such as fluticasone propionate (FP) have dose-related systemic effects, including adrenal suppression. We have therefore investigated the effect of adding a large volume spacer on the systemic bioactivity of FP given via a pressurized metered-dose inhaler (pMDI). Methods: Fourteen healthy volunteers (mean age, 29.9 years old) were studied using an open, randomized, placebo-controlled, three-way crossover design. Single doses of the following were given at 5:00 PM in a randomized sequence: (1) eight puffs of FP by

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pMDI, 1.76 mg (250 micrograms ex-valve, 220 micrograms ex-actuator); (2) eight puffs of FP by pMDI, 250 micrograms, with 750-mL spacer (Volumatic; Allen & Hanburys; Uxbridge, UK); and (3) eight puffs of placebo by pMDI. Measurements were made after each dose, including overnight and early morning urinary cortisol/creatinine ratios and 8:00 AM serum cortisol. Results: Significant ($p < 0.05$) suppression of all three end points occurred with each active treatment compared to treatment with placebo. Furthermore, significant ($p < 0.05$) additional suppression occurred when comparing FP by pMDI alone to FP by pMDI with spacer. Geometric mean fold differences (95% confidence interval for fold difference) between FP by pMDI alone and FP by pMDI with spacer were 1.94-fold (1.00-3.78) for overnight urinary cortisol/creatinine ratio and 1.98-fold (1.26-3.10) for 8:00 AM serum cortisol. This was mirrored by a twofold rise in the number of values for uncorrected overnight urinary cortisol < 10 nmol/10 h: placebo treatment (none of 14 subjects); FP by pMDI (6 of 14 subjects; 43%); and FP by pMDI with spacer (12 of 14 subjects; 86%). Conclusions: The use of a large volume spacer with FP by pMDI results in a twofold increase in the systemic bioavailability as assessed by sensitive measures of adrenal suppression. This, in turn, reflects a twofold improvement in respirable dose delivery with the spacer device.

Effect of Sputum Induction on Spirometric Measurements and Arterial Oxygen Saturation in Asthmatic Patients, Smokers, and Healthy Subjects—Castagnaro A, Chetta A, Foresi A, D'Ippolito R, Malorgio R, Olivieri D. Chest 1999 Oct;116(4):941-945.

Background: Sputum production induced by inhalation of hypertonic saline solution has been proposed as a technique to collect secretions and inflammatory cells from the airways of subjects with bronchial asthma or with a history of smoking. The aim of this study was to determine the effect of a sputum induction procedure on spirometric results and arterial oxygen saturation (S_{aO_2}) in asthmatic patients, smokers, and healthy subjects. Methods: We recruited 14 subjects suffering from asthma (11 men and 3 women; age range, 18 to 49 years), 14 subjects with a history of smoking (5 men and 9 women; age range, 23 to 64 years), and 9 healthy volunteers (7 men and 2 women; age range, 28 to 54 years). To obtain a sample of induced sputum, all subjects inhaled a mist of 3% hypertonic saline solution nebulized for 5 min and repeated the cycle no more than four times. Asthmatic patients were pretreated with 200 micrograms salbutamol (inhaled). During sputum induction, the transcutaneous S_{aO_2} was continuously measured and baseline, fall, and the differences between baseline and fall S_{aO_2} were recorded. Additionally, we measured the duration of mild desaturation (change in $S_{aO_2} < 4\%$) and of marked desaturation (change in $S_{aO_2} > 5\%$) in each subject. Finally, baseline FEV_1 and changes in FEV_1 as a percentage of baseline values were recorded in all subjects. Results: We found that baseline and fall S_{aO_2} values for the three groups were similar. However, in each group a significant mean change in S_{aO_2} was evident during sputum production (asthmatic patients, 6.0%; smokers, 5.3%; healthy subjects, 6.0%). Moreover, the mean durations of mild desaturation were 7 min, 21 s in asthma patients; 8 min, 24 s in smokers; and 7 min, 16 s in healthy subjects. Similarly, the durations of marked desaturation were 1 min, 25 s in asthmatic patients; 1 min, 19 s in smokers; and 1 min, 21 s in healthy subjects. The mean ($\pm$ SD) fall in FEV_1 was not statistically different among the three groups (asthmatic patients, 1.36 $\pm$ 5.6%; smokers, 7.58 $\pm$ 11.76%; and healthy subjects, 0.05 $\pm$ 9.6%). However, one smoker did experience excessive bronchoconstriction (fall in $FEV_1 > 20\%$). Conclusions: This study demonstrated a significant and comparable fall in S_{aO_2} during sputum induction by inhalation of hypertonic saline solution in asthmatic patients, smokers, and healthy subjects. The results suggest that subjects who are hypoxemic before sputum induction require S_{aO_2} monitoring during the procedure.

The Relationships among Pulmonary Function, Aerobic Fitness, and Cognitive Functioning in Older COPD Patients—Etnier J, Johnston R, Dagenbach D, Pollard RJ, Rejeski WJ, Berry M. Chest 1999 Oct;116(4):953-960.

Study objectives: To study the predictive relationships among age, pulmonary function, aerobic fitness, and cognition in people with COPD. Design: Observational study conducted during baseline testing with COPD patients who volunteered to participate in an exercise intervention. Participants: Older adults (age, 56 to 80 years) with COPD. Measurements and results: Age, depression, education level, aerobic fitness, blood oxygen saturation levels, and pulmonary function were assessed. Participants were randomly assigned to take cognitive tests of (1) fluid intelligence, (2) processing speed and working memory span, or (3) processing speed and inhibition. After controlling for education and depression ($F(2,57) = 7.43$; $r^2 = 0.21$), performance on the 6-min walk ($F(1,56) = 15.27$; $r^2 = 0.17$) and age ($F(1,55) = 7.52$; $r^2 = 0.08$) were significant predictors of fluid intelligence. On the speed-of-processing task, performance on the 6-min walk ($F(1,30) = 8.17$; $r^2 = 0.20$), maximum voluntary ventilation ($F(1,29) = 5.81$; $r^2 = 0.16$), and age ($F(1,28) = 5.26$; $r^2 = 0.10$) were significant predictors.

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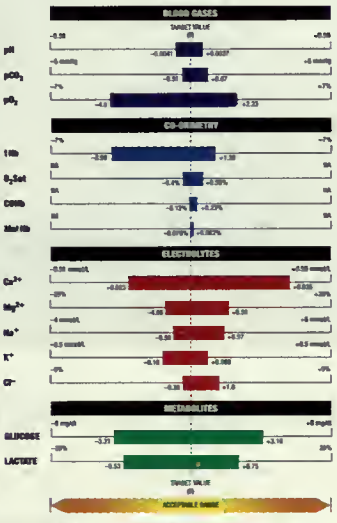

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FVC was a significant predictor ($F(1,25) = 6.37$; $r^2 = 0.18$) of working memory span. The ability to inhibit a response was not significantly predicted by any of the variables assessed. Conclusions: In an older COPD sample, age, aerobic fitness, and pulmonary function are predictive of cognitive performance on various tasks. In particular, age and aerobic fitness are predictive of speed of processing, which is a cognitive variable that may itself underlie performance on a majority of cognitive tasks.

Self-Adjusting Nasal Continuous Positive Airway Pressure Therapy Based on Measurement of Impedance: A Comparison of Two Different Maximum Pressure Levels—Randerath WJ, Parys K, Feldmeyer F, Sanner B, Ruhle KH. Chest 1999 Oct;116(4):991-999.

Study objective: Automatic titration using the forced oscillation technique (FOT) has recently been developed for the treatment of obstructive sleep apnea syndrome (OSAS). So far, it is not known if therapy with automatic nasal continuous positive airway pressure (nCPAP) using a preset upper pressure limitation or a free range (which might lead to higher mean pressure) is preferable with regard to obstructive events, sleep stages, and pressure characteristics. Design: After diagnostic polysomnography, patients were randomly assigned to two settings with the self-adjusting nCPAP (APAP) device based on the FOT. In mode 1, the pressure variation ranged from 4 to 15.5 cm H_2O, and in mode 2, the pressure variation ranged from 4 cm H_2O to an individual upper pressure limit. Patients: Eleven men, aged 53.0 ± 6.8 years with a body mass index of 32.4 ± 5.1 kg/m^2 and an apnea-hypopnea index (AHI) of 31.6 ± 26.6/h. Measurements and results: Manually titrated pressure was at 9.3 ± 2.1 cm H_2O, the mean pressure in mode 1 was 5.4 ± 1.0 cm H_2O ($p < 0.01$), and the mean pressure in mode 2 was 5.1 ± 0.7 cm H_2O ($p < 0.01$). A reduction of respiratory events (baseline AHI, 31.6 ± 26.6/h; AHI in mode 1, 3.4 ± 4.5; AHI in mode 2, 5.0 ± 7.2; each with $p < 0.001$) and an increase in the "rapid eye movement" stage of sleep (baseline, 13.0 ± 5.5%; mode 1, 22.0 ± 7.7 [$p < 0.05$]; mode 2, 23.0 ± 7.9 [$p < 0.01$]) were achieved. In mode 1, the mean pressure was below the manual pressure 91.7 ± 9.3% of the time, and in mode 2, the mean pressure was below the manual pressure 90.4 ± 6.3% of the time. The manual pressure was exceeded by 5.5 ± 7.4% (mode 1) and by 5.2 ± 3.1% (mode 2). Conclusion: We conclude that nCPAP therapy based on the FOT permits the adequate treatment of OSAS with significantly lower pressure than manually titrated nCPAP therapy does. A presetting of an upper pressure limit has no advantage compared to free range.

The Sleep Supine Position Has a Major Effect on Optimal Nasal Continuous Positive Airway Pressure: Relationship with Rapid Eye Movements and Non-Rapid Eye Movements Sleep, Body Mass Index, Respiratory Disturbance Index, and Age—Oksenberg A, Silverberg DS, Arons E, Radwan H. Chest 1999 Oct;116(4):1000-1006.

Study objectives: To evaluate the impact of sleep position on optimal nasal continuous positive airway pressure (nCPAP [op-nCPAP]) in obstructive sleep apnea (OSA) patients and to investigate how rapid eye movements (REM) and Non-REM (NREM) sleep, body mass index (BMI), respiratory disturbance index (RDI), and age are related to this effect. Design: Retrospective analysis. Setting: Sleep Disorders Unit at Loewenstein Hospital Rehabilitation Center. Patients: Eighty-three consecutive adult OSA patients who underwent a complete nCPAP titration. From this group, 60 patients who spent at least 30 min in both the supine (Sup) and lateral (Lat) positions and 46 patients who had data on both positions during REM and NREM sleep were included in the analysis. Results: In most OSA patients (52; 86.7%), the recommended op-nCPAP was obtained when the patients slept in the Sup posture. The mean op-nCPAP was significantly higher in the Sup posture (10.00 ± 2.20 cm H_2O) than it was in the Lat posture (7.61 ± 2.69 cm H_2O). The op-nCPAP was significantly higher in the Sup position than it was in the Lat position in both REM and NREM sleep, as well as in the severe BMI group (BMI ≥ 30) and in the less obese group (BMI < 30). Similarly, in the severe (RDI ≥ 40) and less severe groups (RDI < 40), as well as in both age groups (< and > 60 years of age), the op-nCPAP was significantly higher in the Sup posture than it was in the Lat posture. Irrespective of the four parameters mentioned, the actual differences in op-nCPAP between the two body postures were almost identical, ranging between 2.31 and 2.66 cm H_2O. Conclusions: For most OSA patients, the op-nCPAP level is significantly higher in the Sup position than it is in the Lat position. This is true for REM and NREM sleep, for obese and nonobese patients, for patients with different degrees of severity, and for young and old OSA patients. Since the op-nCPAP was highest in the Sup posture during REM sleep, no nCPAP titration should be considered complete without the patient having slept in the Sup posture during REM sleep.

Differences in the Response Times of Pages Originating from the ICU—Moss M, Trow TK, Clardy N. Chest 1999 Oct;116(4):1019-1024.

Study objectives: To determine whether the type of paging system causes significant differences in the response time by physicians to their pages in an ICU setting. Design and setting: Prospective cohort study performed in the ICU of two university-affiliated hospitals. All pages were classified by several different variables, including the type of paging system: direct paging if a nurse or hospital operator could directly place the page, or indirect paging if a nurse or hospital operator was required to contact the physician's office or a private answering service who would then independently contact the physician. The main outcome measure was physicians' response time, in minutes, to pages originating from the ICU. Results: During a 100-day period, 402 pages were sent and answered by 166 different physicians (87 attending physicians and 79 housestaff/physician assistants). The median response time for all pages was 3 min with a 25 to 75% quartile of 1 to 8 min. Twenty-five percent of the pages placed through an indirect system were associated with a response time of ≥ 29 min. In a multivariate model with the response time dichotomized at ≥ 15 min ("slow") or < 15 min ("adequate"), pages placed through an indirect system were answered significantly more slowly than pages placed through a direct system ($p < 0.001$; odds ratio, 4.36; 95% confidence interval, 2.05 to 9.29). Pages answered in an adequate amount of time were also associated with a significantly higher degree of overall nursing satisfaction with the care delivered by the physician in response to the specific page when compared with pages answered in a "slow" manner ($p < 0.001$). Conclusions: Physicians who use an indirect paging system are significantly slower in their response to ICU pages when compared with physicians who utilize a direct paging system. These results may lead to improvements in paging systems used by physicians who care for patients in an ICU setting.

Reengineering Respiratory Support Following Extubation: Avoidance of Critical Care Unit Costs—Munshi IA, DeHaven B, Kirton O, Sleeman D, Navarro M. Chest 1999 Oct; 116(4):1025-1028.

Study objective: We prospectively investigated alternative clinical practice strategies for critically ill trauma patients following extubation to evaluate the cost-effectiveness of these maneuvers. The primary change was elimination of the routine use of postextubation supplemental oxygen, with concurrent utilization of noninvasive positive pressure ventilatory support (NPPV) to manage occurrences of postextubation hypoxemia. Design: Prospective, consecutive accrual of patients undergoing extubation. Setting: Trauma ICU in a university hospital. Interventions and measurements: All patients received mechanical ventilation using pressure support ventilation (PSV) with continuous positive airway pressure (CPAP) as the primary mode. The patients were extubated to room air following a 20-min preextubation trial of 5 cm H_2O CPAP at F_{IO_2} of 0.21, and demonstrating a spontaneous respiratory rate ≤ 38 breaths/min,

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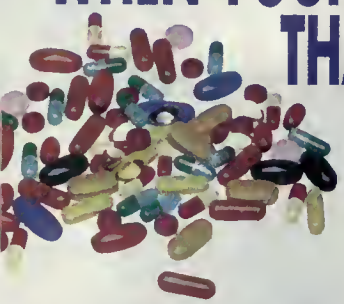

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Reviews the pharmacology section of the revised NAEPP Guidelines. Additionally, the potential hazards and undesirable side effects of commonly prescribed medications will be discussed, with alternative treatment regimens suggested. Featuring William Luskin, MD, and David J. Pierson, MD. 80-min. videotape.

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pH ≥ 7.30, P_{aCO_2} ≤ 50 mm Hg, and P_{aO_2} ≥ 50 mm Hg. The subgroup of patients who became hypoxemic (pulse oximetric saturation < 88%) within 24 h of extubation were treated with NPPV for up to 48 h duration. Patients who failed NPPV were reintubated. Four hundred fifty-one (84%) patients were successfully extubated to room air. Seventy-two patients (13%) became hypoxemic within 24 h, and NPPV was administered. Fifty-two patients (72% of those who were hypoxemic) responded to NPPV, while 20 patients failed to respond to therapy, were reintubated, and received mechanical ventilation for a mean of 4 days. Thirteen additional patients (2%) were reintubated for reasons other than hypoxemia. The overall reintubation rate for the group (n = 536) was 6.2%; for the postextubation hypoxemic group who failed NPPV, the reintubation rate was 3.7%. The elimination of routine supplemental oxygen via nasal cannula following extubation resulted in a potential direct cost avoidance of $50,006.88 for 451 patient days. Moreover, the 52 patients who were spared reintubation and mechanical ventilation provided an additional potential cost avoidance of $19,740.24 in unused ventilator days per patient. Conclusion: Eliminating the routine use of supplemental oxygen and employing NPPV as a method to prevent reintubation can facilitate a more aggressive, cost-effective strategy for the management of the trauma ICU patient who has been extubated.

The Effect of Varying Inspiratory to Expiratory Ratio on Gas Exchange in Partial Liquid Ventilation—Lim CM, Koh Y, Shim TS, Lee SD, Kim WS, Kim DS, Kim WD. Chest 1999 Oct;116(4):1032-1038.

Background: In partial liquid ventilation (PLV), the nondependent lung was observed to be inflated first and the dependent lung later. The inflational time difference between the lung regions can lead to maldistribution of tidal gas and inefficient gas bubbling in the slow-inflating region during PLV. In this situation, increasing the inspiratory to expiratory (I:E) ratio of the mechanical ventilator would lessen the heterogeneity of regional ventilation and improve gas exchange possibly to a greater degree than in gas ventilation (GV). Design and setting: Animal study at the Asan Institute for Life Sciences, Seoul, Korea Subjects: Eighteen rabbits (2.6 ± 0.5 kg) with acute lung injury by saline solution lavage. Interventions: Three I:E ratios were tried in GV and then in PLV. I:E ratios were changed by adjusting pause (1:2, 1:1, and 2:1; group 1) or by adjusting inspiratory flow rate (1:3, 1:1, and 2:1; group 2). Measurements and results: With increasing I:E ratio in all animals, P_{aO_2}/F_{IO_2} increased (80 ± 24, 143 ± 74, and 147 ± 88 mm Hg; p = 0.001), and P_{aCO_2} decreased (74 ± 15, 66 ± 16, and 66 ± 15 mm Hg; p = 0.006). The increases of P_{aO_2}/F_{IO_2} from 1:2/1:3 to 1:1 (p = 0.006) and from 1:1 to 2:1 (p = 0.036) were both greater in group 1 than in group 2. P_{aCO_2} decreased with increasing I:E ratio in group 1, but not in group 2. The change of P_{aO_2}/F_{IO_2} by varying the I:E ratio was 49 ± 65% in PLV and 14 ± 14% in GV (p = 0.003). Conclusions: Extending the I:E ratio, especially by adding pause, improved gas exchange in PLV. Oxygenation in PLV was affected by the I:E ratio to a greater degree than in GV.

Guidelines in Pulmonary Medicine: A 25-Year Profile—Hackner D, Tu G, Weingarten S, Mohsenifar Z. Chest 1999 Oct;116(4):1046-1062.

Objective: We attempted to identify clinical practice guideline and pathway articles in the area of pulmonary medicine published in peer-reviewed journals since 1974. Design: Review. Data sources: MEDLINE, the Cochrane Database, Best Evidence, and Abstracts of Clinical Care Guidelines from January 1974 to December 1998. Study selection: All articles contained relevant search terms for pulmonary topics and were included irrespective of setting (primary or specialty, inpatient or outpatient). Controlled and uncontrolled trials as well as observational studies and consensus opinion/statements were all identified. The articles were stratified by design as well as by pulmonary topic. Data extraction: Limited data on study type, study focus, year of publication, and results of study were abstracted. Results: Our criteria yielded 271 articles, including 115 consensus statements and expert opinion guidelines; 30 controlled studies, meta-analyses, or systematic reviews; and 126 uncontrolled trials and observational studies. Of these, 82 articles (30.3%) related to asthma, 46 articles (17.0%) related to COPD, and 36 articles (13.3%) related to pneumonia. In addition, we tracked the increasing publication of all guideline-related pulmonary articles; randomized, controlled trials (RCTs); systematic reviews; and consensus statements by year for the past 25 years. Conclusion: Pulmonary guidelines are increasingly published in peer-reviewed journals, but few are tested clinically in RCTs. There is continued reliance on consensus statements and expert opinion. Pulmonary guideline publications have continued to dramatically increase in number and in importance since 1974, both on the local level and internationally.

Pulmonary Artery Catheterization and Esophageal Doppler Monitoring in the ICU—Marik PE. Chest 1999 Oct;116(4):1085-1091.

The clinical assessment of cardiac performance and ventricular preload is notoriously unreliable in critically ill patients. Consequently, a number of technologies have been developed to provide the clinician with indexes of cardiovascular function to assist in therapeutic decision making. Foremost among these is the pulmonary artery catheter (PAC). Indeed, the PAC has largely shaped the practice of modern critical care. Yet, the information provided by the PAC is largely misunderstood, and its efficacy is never proven. Recently, continuous esophageal Doppler monitoring has emerged as an alternative to pulmonary artery catheterization. This paper evaluates the clinical utility of the PAC and esophageal Doppler monitoring in assessing the hemodynamic status of ICU patients.

Dyspnea Differentiation Index: A New Method for the Rapid Separation of Cardiac vs Pulmonary Dyspnea—Ailani RK, Ravakhah K, DiGiovine B, Jacobsen G, Tun T, Epstein D, West BC. Chest 1999 Oct;116(4):1100-1104.

Study objective: To assess the utility of a new parameter in the differentiation of dyspnea of cardiac origin from dyspnea of pulmonary origin. Methods: The peak expiratory flow (PEF) rate and the partial pressure of oxygen in arterial blood (P_{aO_2}) were measured in 71 patients with the chief complaint of dyspnea. The patients were treated in the hospital, and the final diagnosis (cardiac or pulmonary) of the cause of dyspnea was made at discharge. We defined a new measure, the dyspnea differentiation index (DDI), as (PEF × P_{aO_2})/1,000. We performed a receiver operating characteristic (ROC) curve analysis of the data to define the measure that best distinguished cardiac from pulmonary dyspnea. The curves also allowed us to establish an optimal cut-off point to distinguish between cardiac and pulmonary dyspnea. Results: Patients with pulmonary dyspnea had a significantly lower mean PEF than patients with cardiac dyspnea (144 ± 66 vs 267 ± 97 L/min, respectively; p < 0.001). They also had a lower DDI than patients with cardiac dyspnea (8.4 ± 4.0 vs 18.4 ± 7.9 L-mm/min, respectively; p < 0.001). These two measures, PEF and DDI, also best distinguished pulmonary from cardiac dyspnea. PEF was able to diagnose the correct cause of dyspnea in 72% of patients, and DDI was correct in 79% of patients. This compares favorably to the performance of the emergency department physicians, who were able to predict the correct diagnosis in only 69% of patients. Conclusion: These results demonstrate that the PEF by itself is useful in differentiating between cardiac and pulmonary causes of dyspnea, but that the calculation of DDI is superior in this regard.

Evaluation of Bronchodilator Response in Patients with Airway Obstruction—Quadrelli SA, Roncoroni AJ, Montiel GC. Respir Med 1999 Sep;93(9):630-636.

The aim of this study was to define the most useful index of expressing bronchodilator response and to distinguish between asthma and

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Tools of the Trade

Interested in the long-term oxygen therapy topics discussed in this issue? Here are some resources you may find helpful.

Basic Oxygen Administration

Presents the primary goals of oxygen therapy and the clinical conditions that warrant oxygen administration. Also covers oxygen sampling techniques for various types of patients including newborns and various oxygen administration appliances. Includes oxygen administration hazards, assessment factors for evaluation of oxygen delivery, the principles of humidification and factors affecting efficiency of devices, proper attachment of oxygen devices, and trouble-shooting of bubble humidifiers. Software, 3 1/2" Disk. Requires Windows® 3.1 or higher.
Item PEL3 $65.00

Hemodynamics

Presents the basic concepts involved in monitoring and interpretation of hemodynamic values. It includes general guidelines for use and maintenance of lines, systemic blood pressure including central venous pressure, cardiac output, oxygen content, Swan-Ganz pulmonary artery catheter measurements, clinical applications of hemodynamic measurements, relationship between clinical conditions and hemodynamic measurements, and case studies which consist of 20 questions on clinical conditions, with associated laboratory and hemodynamic values. Software, 3 1/2" Disk. Requires Windows® 3.1 or higher.
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Airway Management: Tricks of the Trade

Learn how to manage the obstructed airway and how to determine when an artificial airway is necessary. Also presented are the methods of assessing endotracheal tube positioning, monitoring the intubated patient, and determining when extubation is feasible. Featuring Charles G. Durbin, Jr., MD, FCCM, and David J. Pierson, MD. 80-minute videotape.
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Hemodynamic Monitoring

Identifies the different forms of invasive and noninvasive hemodynamic monitoring, their main complications, and appropriate levels. Discusses how positive end-expiratory pressure and other therapies affect parameters. Featuring John J. Marini, MD, and David J. Pierson, MD. 80-minute videotape.
Item VC22 $49.95 ($99.00 nonmembers)

Blood Gas and Related Measurements: Laboratory Versus Bedside Devices

Presents the various means of obtaining blood gas information using invasive and noninvasive devices, the need for such devices, and when they should be used. Featuring Barry R. Shapiro, MD, and Richard D. Branson, BS, RRT. 80-minute videotape.
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An Introduction to Hemodynamic Monitoring

Teaches you to recognize the types of equipment used for hemodynamic measurements. It also instructs in position of the pressure transducer, identification of pressure waveforms, and measurement of cardiac output. Discusses anatomic normal pressure values, oxygen saturation values, and the two major determinants of cardiac output. Individual Independent Study Package (IISP).
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Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation

After completing this IISP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy. Individual Independent Study Package (IISP).
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Tracheal Intubation II: Routes of Intubation

Describes the four routes of tracheal intubation and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation. Individual Independent Study Package (IISP).
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Arterial Blood Gas Interpretation

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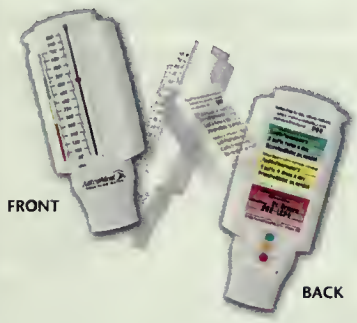

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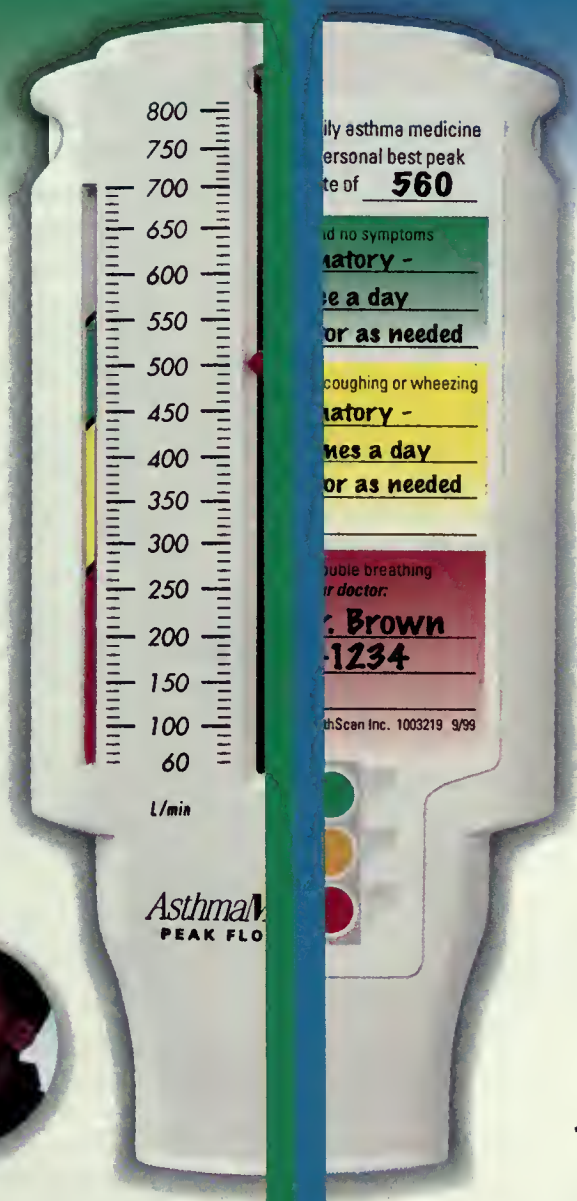

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Reference: 1. National Asthma Education and Prevention Program: Expert Panel Report II: Guidelines for the Diagnosis and Management of Asthma. Bethesda, MD: U.S. Department of Health and Human Services; 1997.

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COPD. A prospective study was carried out of bronchodilator response in 142 asthmatics and 58 COPD patients in a university hospital. Reversibility was expressed as: 1. absolute change (Δ abs); 2. % of initial (Δ %init); 3. % of predicted (Δ %pred) and 4. % of maximum possible response (Δ %max). Dependence on forced expirations volume in 1 sec (FEV_1) as % of predicted and sensitivity and specificity for diagnosis of asthma were established. A relationship between Δ abs and initial FEV_1 was not found in asthma (Δ abs vs. % initial FEV_1, r = 0.07) or COPD (r = 0.02). Δ %pred did not show a correlation in asthma (r = 0.10) or COPD (r = 0.06). Δ %init was dependent on the baseline value in asthma (r = 0.38, $p \leq 0.001$) but not in COPD (r = 0.18, p = n.s.). Δ max was dependent in both. The combination of best sensitivity and specificity to separate asthma and COPD was obtained with Δ abs (70.4 or 70.6%). The worst specificity for asthma diagnosis was obtained with Δ %init (50%). The best likelihood ratios were obtained with Δ abs and Δ %pred and the worst likelihood ratio with Δ %init. Δ %init is not recommended as an index for differential diagnosis between asthma and COPD; 2) Δ %init overscores bronchodilator response in patients with low FEV_1. The independence of each bronchodilator response index should be verified in clinical trials for each selected sample.

Volume Calibration Alone May Be Misleading—van den Boom G, van der Star LM, Folgering H, van Schayck CP, van Weel C. Respir Med 1999 Sep;93(9):643-647.

The use of spirometry is becoming more and more widespread in non-laboratory situations such as general practice or occupational medicine. In these non-laboratory situations, volume calibration with a 3000 mL syringe is often the only feasible method to ensure that the spirometer produces valid and reproducible data. Sophisticated equipment to calibrate forced manoeuvres with standard waveforms are not present. In this study, we assessed whether volumetric calibration is a guarantee for valid and comparable spirometric results. Two portable spirometers were tested. On 8 consecutive test days, both spirometers were calibrated with a 3000 mL syringe in accordance with the American Thoracic Society (ATS) guidelines. The comparability of the spirometric results (forced expiratory volume in 1 S, FEV_1) was tested in two ways. Firstly, the spirometers were compared to each other using the results from 43 volunteers on the same 8 test days. The spirometers were presented in a randomized order and volunteers were asked to perform a series of reproducible manoeuvres in both spirometers. Paired observations were analysed, using Bland and Altman plots. Secondly, the spirometers were compared to a 'gold standard', a computer-driven syringe (CDS). Calibration with the 3000 mL syringe showed that both spirometers complied with the ATS criteria for volume calibration for diagnostic spirometry. However, paired FEV_1 data obtained in subjects showed a systematic, volume-dependent difference between the two spirometers (mean difference: 289 mL, $p < 0.001$, systematic difference: 8.6%, $p < 0.0001$). This systematic difference was confirmed by the comparisons with the CDS. Volume calibration may be misleading. The results from volume calibration may meet the ATS criteria, but this is no guarantee that data from forced manoeuvres are accurate. If CDS equipment to simulate standard wave forms is not available, it is recommended that biological calibration is performed regularly and, if possible, that paired data from two (or more) different spirometers are compared.

Patterns of Medical Practice in Cystic Fibrosis: Part 1. Evaluation and Monitoring of Health Status of Patients—Konstan MW, Butler SM, Schidlow DV, Morgan WJ, Julius JR. Pediatr Pulmonol 1999 Oct;28(4):242-247.

This report characterizes patterns of evaluation and monitoring of the health status of patients with cystic fibrosis (CF) as observed in the Epidemiologic Study of Cystic Fibrosis (ESCF), and compares these practices to published guidelines. All patients (18,411) who enrolled in ESCF at 194 study sites in the United States and Canada from December 1, 1993 to December 31, 1995 were considered for study. Patients enrolled before January 1, 1995 with ≥1 healthcare encounters during 1995 (12,631) were included in the analysis. Patients enrolled after January 1, 1995 (5,266), or who died (354), withdrew from the study (128), or were lost to follow-up (21) were excluded. Frequency of encounters (outpatient and hospital), spirometry, respiratory tract cultures, and chest radiographs were recorded during a 1-year period (1995) and analyzed by gender, age, severity of lung disease, and presence of any Pseudomonas species in the respiratory tract. The 12,631 patients had 53,024 outpatient visits. In 57.5% of patients, the recommended criteria of ≥4 total visits per year were met. Only 27.4% of all patients had ≥4 routine visits; 3.1% had only sick visits, and 59.0% had no sick visits. One third (34.6%) were hospitalized at least once, for a total of 8,561 hospitalizations. Older patients with lower pulmonary function and Pseudomonas in their respiratory tract had fewer routine visits and more sick visits, and were hospitalized more than were younger patients. In three fourths (75.8%) of patients the recommended criterion of two spirometry assessments per year was met, whereas in 79.3% the criterion of one culture was met, and in 68.3% the criterion of one radiograph/year was met. We conclude that in the majority of CF patients, the recommended criteria for routine evaluation and monitoring were met. However, in a rather substantial number they were not. An increase in the utilization of healthcare resources was observed in patients with more severe disease. This information will help to establish benchmarks for future quality assessment programs.

Why Don't Physicians Follow Clinical Practice Guidelines? A Framework for Improvement—Cabana MD, Rand CS, Powe NR, Wu AW, Wilson MH, Abboud PA, Rubin HR. JAMA 1999 Oct 20;282(15):1458-1465.

CONTEXT: Despite wide promulgation, clinical practice guidelines have had limited effect on changing physician behavior. Little is known about the process and factors involved in changing physician practices in response to guidelines. OBJECTIVE: To review barriers to physician adherence to clinical practice guidelines. DATA SOURCES: We searched the MEDLINE, Educational Resources Information Center (ERIC), and HealthSTAR databases (January 1966 to January 1998); bibliographies; textbooks on health behavior or public health; and references supplied by experts to find English-language article titles that describe barriers to guideline adherence. STUDY SELECTION: Of 5658 articles initially identified, we selected 76 published studies describing at least 1 barrier to adherence to clinical practice guidelines, practice parameters, clinical policies, or national consensus statements. One investigator screened titles to identify candidate articles, then 2 investigators independently reviewed the texts to exclude articles that did not match the criteria. Differences were resolved by consensus with a third investigator. DATA EXTRACTION: Two investigators organized barriers to adherence into a framework according to their effect on physician knowledge, attitudes, or behavior. This organization was validated by 3 additional investigators. DATA SYNTHESIS: The 76 articles included 120 different surveys investigating 293 potential barriers to physician guideline adherence, including awareness (n = 46), familiarity (n = 31), agreement (n = 33), self-efficacy (n = 19), outcome expectancy (n = 8), ability to overcome the inertia of previous practice (n = 14), and absence of external barriers to perform recommendations (n = 34). The majority of surveys (70 [58%] of 120) examined only 1 type of barrier. CONCLUSIONS: Studies on improving physician guideline adherence may not be generalizable, since barriers in one setting may not be present in another. Our review offers a differential diagnosis for why physicians do not follow practice guidelines, as well as a rational approach toward improving guideline adherence and a framework for future research.

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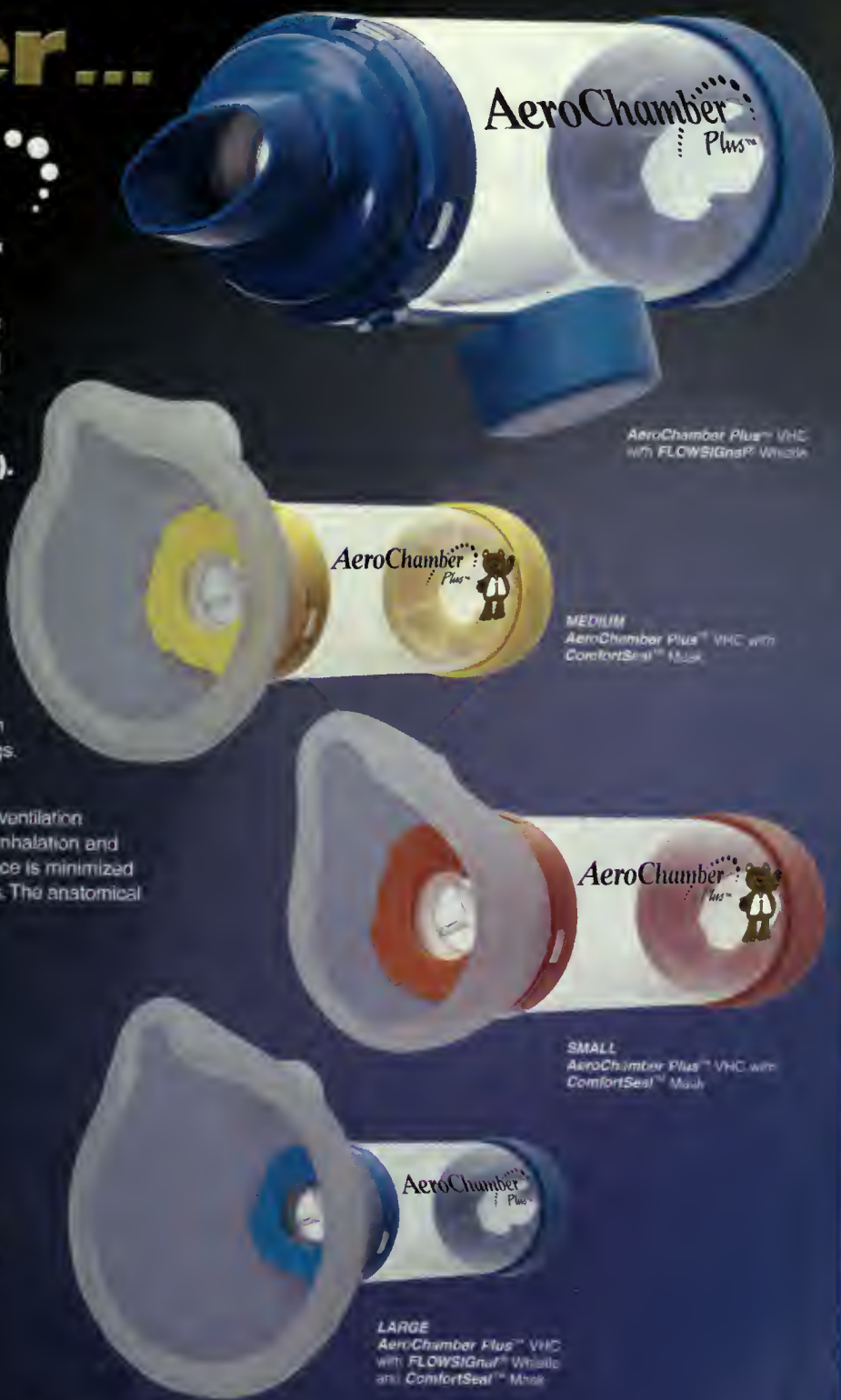

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Long-Term Oxygen Therapy

PART II OF
TWO SPECIAL ISSUES

Containing the manuscripts and discussions from the State-of-the-Art Conference held August 6-8, 1999, in Cancún, Mexico.

This Conference was supported by
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Long-Term Oxygen Therapy in Conditions Other Than Chronic Obstructive Pulmonary Disease

Jan Zieliński MD

Introduction
Sequelae of Tuberculosis
Interstitial Pulmonary Fibrosis
Kyphoscoliosis
Cystic Fibrosis
Summary

[Respir Care 2000;45(2):172–176] *Key words: long-term oxygen therapy, tuberculosis, interstitial pulmonary fibrosis, kyphoscoliosis, cystic fibrosis.*

Introduction

More than one million patients around the world receive long-term oxygen therapy (LTOT). The majority of these patients have chronic obstructive pulmonary disease (COPD). The literature concerning indications for and results of LTOT in COPD patients is rather extensive. Much less has been written about the use of LTOT in patients with other respiratory diseases. In fact, no large controlled investigations have been published concerning the effects of LTOT in non-COPD patients, like the British Medical Research Council (MRC) study[1] and the Nocturnal Oxygen Therapy Trial (NOTT)[2] study did for COPD.

Table 1 shows the percentages of non-COPD patients receiving LTOT in several countries. These range from 61% in Japan[3] to 6.6% in the Czech Republic.[6] In the United States, Medicare beneficiaries represent 80% of all LTOT use, of which 76% was prescribed for COPD. The most frequent non-COPD indication was heart failure.[5]

The two most frequent non-COPD diseases treated with LTOT are sequelae of tuberculosis (TB) and interstitial pulmonary fibrosis (IPF). Next to those are pneumoconiosis, kyphoscoliosis, and bronchiectasis. Table 2 shows those diseases' proportional contributions in several countries. Table 3 lists non-COPD lung diseases in which LTOT is prescribed.

Children constitute a specific group of patients who receive LTOT exclusively for diseases other than COPD. In a group of 287 children treated for chronic respiratory failure with LTOT, mechanical ventilation, or both, more than half presented with restrictive lung disease. The majority of them suffered from neuromuscular disease. The rest were treated for obstructive pulmonary disease: bronchopulmonary dysplasia or cystic fibrosis (CF).[10]

Sequelae of Tuberculosis

In many countries patients with lungs destroyed by TB form the second largest LTOT group after COPD.[3,4,7,8] This group of patients encompasses a variety of different forms, such as fibrocavitary lesions affecting large parts of the lungs, patients post thoracoplasty and lung resection, and patients with fibrothorax after artificial pneumothoraces or pleural effusions. Quite a number of those patients also suffer from smoking-related COPD.[3,4,7,8] High venous admixture is typical for such a group.

Life expectancy with LTOT was invariably found to be better in patients with sequelae of TB than in patients with COPD.[3,4,7,8] The main reason for this finding is stability and lack of progression of the underlying disease. Younger age, female sex, good nutritional status, and higher arterial carbon dioxide tension (P_{aCO_2}) were found to be positive prognostic factors.[4] Also, Aida et al[11] followed a group of more than 3,000 patients with TB sequelae and found that hypercapnia ($P_{aCO_2} \geq 45$ mm Hg) favorably influenced

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Table 1. Relation (in Percent) of COPD to Other Diagnoses Treated with LTOT in Several Countries

	Japan[3]	France[4]	United States[5]	Czech Republic[6]	Poland[7]	Sweden[8]	Netherlands[9]
Total Number of patients	31,621	26,140	8,418	1,296	1,000	698	528
% COPD	39	58.7	76	93.4	67.6	59.9	70
% Other	61	41.3	24	6.6	32.4	40.1	30

COPD = chronic obstructive pulmonary disease.
LTOT = long-term oxygen therapy.

Table 2. Most Frequent Diseases Treated with LTOT

Diagnosis	Japan[3]	France[4]	Poland[7]	Sweden[8]	Netherlands[9]
TB sequelae	24.8	15.9	8.8	4.7	—
IPF	9.3	9.6	8.6	8.3	2
Pneumoconiosis	3.3	3.5	0.4	—	6
Kyphoscoliosis	—	6	2.9	1.1	3
Bronchiectasis	3.7	6	2.9	—	—

LTOT = long-term oxygen therapy.
TB = tuberculosis.
IPF = interstitial pulmonary fibrosis.

Table 3. Pulmonary Diseases Other Than COPD in Which LTOT Is Prescribed

Interstitial pulmonary fibrosis
Tuberculosis sequelae
Pneumoconiosis
Kyphoscoliosis
Neuromuscular diseases
Bronchiectasis
Pulmonary granulomatoses
Cystic fibrosis
Obstructive sleep apnea syndrome with concomitant COPD (overlap syndrome)
Severe pulmonary hypertension:
primary
thromboembolic

COPD = chronic obstructive pulmonary disease.
LTOT = long-term oxygen therapy.

survival. Taking risk of death as 1 in normocapnic patients, the relative risk of mortality was 0.76 in patients with P_{aCO_2} between 45 and 55 mm Hg, 0.64 in patients with P_{aCO_2} between 55 and 65 mm Hg, and 0.49 in patients with $P_{aCO_2} > 65$ mm Hg. Those findings applied only to patients with no significant concomitant COPD. Interestingly, patients with hypercapnia were significantly older and more hypoxemic than normocapnic patients. Also, Ström et al[12] found an independent positive effect of hypercapnia on survival in patients with old sequelae of TB.

In the coming decade there will be a decrease in the number of patients with sequelae of TB referred for LTOT, thanks to the availability, for many years, of effective anti-TB treatment. In Sweden, a retrospective analysis of patients receiving LTOT before 1988 indicated that 18% of the total number were TB sequelae patients.[8] In contrast, among patients starting LTOT between 1989 and 1992 only 4.7% had the TB sequelae diagnosis.[13]

Interstitial Pulmonary Fibrosis

Hypoxemia in COPD patients is caused by ventilation-perfusion inequality, venous admixture, and diffusion impairment. In the early stage of the disease, hypoxemia at rest is rather mild, with abrupt aggravation on exercise. Fibrosis of the interstitium leads to progressive closure of the pulmonary vascular bed and to pulmonary hypertension. It seems that alveolar hypoxia plays a minimal role in the development of pulmonary hypertension in IPF patients. There are no longitudinal data on effects of LTOT on pulmonary hypertension in IPF patients.

In 1988, the Clinica del Lavoro Foundation of Italy promoted an international, multicenter, controlled study on the effects of LTOT on pulmonary hemodynamics and survival in IPF patients.[14] I participated in that study, which was completed after 5 years, but the results have not yet been published. The initial data and survival curves for treated and control patients were kindly given to me by Dr Alberto Braghiroli, the principal investigator.

Patients of both sexes, under 79 years of age, affected by IPF not previously treated with oxygen were selected according to the following criteria:

1. Total lung capacity < 80% of the predicted plethysmographic value.

2. Arterial partial pressure of oxygen (P_{aO_2}) between 45 and 60 mm Hg on 4 consecutive weekly determinations.

The group collected 62 patients, 49 with idiopathic pulmonary fibrosis and 13 with other forms of fibrosis. The mean age was 62 ± 7 years. The male to female ratio was 5:4. Thirty-seven patients were allocated to an oxygen-treated group and 25 served as controls. The initial pulmonary function tests obtained the following values. Mean

vital capacity was 51 ± 13% of predicted. The ratio of the forced expiratory volume in the first second to the vital capacity was 103 ± 12% of predicted. Total lung capacity was 65 ± 18% of predicted. The diffusing capacity of carbon monoxide was 43 ± 10% of predicted. Mean P_{aO_2} was 54 ± 10 mm Hg and mean P_{aCO_2} was 36 ± 5 mm Hg.

Pulmonary artery catheterization was performed using a Swan-Ganz thermodilution catheter. After initial measurements were taken, patients received oxygen at a flow of ≥ 2 L/min to increase P_{aO_2} to > 65 mm Hg. Second measurements were taken after one hour of oxygen. The results are shown in Table 4.

Patients presented with moderate pulmonary hypertension, and mean pulmonary arterial pressure was 33 ± 11 mm Hg. Administration of oxygen increased P_{aO_2} from 54 ± 10 mm Hg to 79 ± 23 mm Hg. No pulmonary artery vasodilation was observed, and pulmonary arterial pressure and pulmonary vascular resistance were unchanged.

Patients were followed for 3 years. The survival rate was very low: only 20% survived 3 years. There was no difference in the survival rate between controls and patients treated with oxygen.

A very poor prognosis in IPF patients despite LTOT was also found by other groups. Crockett et al[15] made a retrospective analysis of a group of IPF patients receiving LTOT. Fifty percent survived 1 year. Only 10% survived 3 years. Ström et al[13] reported a 20% survival rate at 3 years in IPF patients receiving LTOT. The ANTADIR group[4] followed more than 1,300 IPF patients and found a median survival time of 15 months.

In Poland we investigated 81 IPF patients, 61 females and 21 males with a mean age of 48 ± 13 years. They presented with severe ventilatory restriction, mean vital capacity of 1.59 ± 0.67 L (45% of predicted), and forced expiratory volume in the first second of 1.17 ± 0.54 L. Mean P_{aO_2} while breathing air was 51 ± 8 mm Hg, and rose to 65 ± 10 mm Hg while breathing oxygen. The group started LTOT and was followed for 3 years. The survival rate at 3 years was 24%. Death rate was similar in both sexes up to 1.5 years. After that time, survival in women was better than in men. Only female sex and better spirometric values correlated with better prognosis. Neither age nor P_{aO_2} while breathing air or breathing oxygen had a significant effect on survival.

Table 4. Pulmonary Haemodynamics in tPF Patients Breathing Room Air and Breathing Supplemental Oxygen

Variable	Room Air	Oxygen
PAP (mm Hg)	33 ± 11	33 ± 9
CO (L/min)	4.47 ± 1.22	3.62 ± 0.67
PVR (mm Hg/L/min)	3.55 ± 2.2	3.83 ± 1.9
P_{aO_2} (mm Hg)	54 ± 10	79 ± 23*

IPF = interstitial pulmonary fibrosis.
PAP = pulmonary artery pressure (mean).
CO = cardiac output.
PVR = pulmonary vascular resistance.
P_{aO_2} = arterial oxygen tension.
*$p < 0.01$.

Ström and Boman[16] also found that in IPF patients spirometric values (and not blood gases) correlated with survival.

Kyphoscoliosis

Kyphoscoliosis and other thoracic spine deformities may lead to important blood gas exchange abnormalities. Alveolar hypoventilation and ventilation-perfusion mismatching may result in hypoxemia, hypercapnia, and development of pulmonary hypertension. Increased elastic resistance to breathing leads to rapid shallow breathing and an increased ratio of dead space to tidal volume. Although noninvasive positive-pressure ventilation with supplemental oxygen seems to be the treatment of choice in patients with kyphoscoliosis, many patients receive LTOT alone.

In the largest series published so far, concerning 912 patients with kyphoscoliosis treated by ANTADIR in France,[4] more than 40% survived 9 years. Ström et al[17] followed 80 kyphoscoliotics. Female sex, younger age, higher body weight, and higher P_{aO_2} and P_{aCO_2} were independent favorable prognostic factors. More than 70% of patients survived 3 years. These Swedish authors also found that hypercapnia was not an ominous prognostic factor in kyphoscoliotics, as it was found to be for COPD patients.[1]

In assessing the results of LTOT in kyphoscoliosis patients, one has to consider that many patients have also been treated with noninvasive or invasive positive-pressure ventilation.[4,17]

Tobiasz et al[18] also found good prognosis for kyphoscoliosis patients. Seventy percent of patients survived 3 years. Good prognosis in kyphoscoliosis may result from the fact that thoracic spine deformity is a very slowly progressive disease. Also, kyphoscoliotics are younger than patients with other diagnoses at the start of LTOT.

Cystic Fibrosis

CF is apparently the only non-COPD disease in which oxygen use has been studied in a controlled manner. Zinman et al[19] did not find benefits from nocturnal oxygen supplementation in children or young adults with CF. The oxygen treatment did not affect mortality rate, hospitalizations, or progression of the disease over a 3-year period. However, school and work attendance were better in the oxygen-treated patients than in the controls.

Zinman's study[19] had a relatively small number of patients (28 in both arms) and the design of the study may

have affected the observed outcome. Although nocturnal oxygen supplementation improves sleep quality in CF patients, perhaps it would be more beneficial to treat such patients with ambulatory oxygen during the day. Because of their young age, CF patients are physically very active. Relief of hypoxic pulmonary hypertension and tissue hypoxia during exercise might result in better survival in oxygen-treated patients.

Nixon et al[20] confirmed beneficial effects of oxygen during exercise in CF patients. Thirty-six CF patients were subjected to two exercise tests, one in normoxia, the other while breathing 30% oxygen. Oxygen supplementation decreased ventilatory and cardiovascular work at a given work load. In a study by Marcus et al,[21] oxygen supplementation increased exercise capacity and reduced desaturation of arterial blood at peak exercise.

Summary

Prescriptions of LTOT in non-COPD diseases vary from 20% to 60% of the total number of patients profiting from this form of treatment. It seems that in countries where the percentage of non-COPD indications is still high, the proportions will change toward higher use of LTOT in COPD patients in the near future.

There is a dearth of controlled studies evaluating the effects of LTOT in non-COPD diseases. There are at least two important factors preventing realization of such studies. First, non-COPD indications for LTOT encompass a large number of diseases. Each of them should be separately studied because of differences in the pathophysiological mechanisms and natural history of the diseases. Small numbers of patients with a specific diagnosis would require multicenter, international studies, which are difficult to perform in a rigorous manner.

A more important handicap is ethical reasons. Many years ago LTOT was approved as a routine method of treatment for severe stable hypoxemia. It would be difficult to obtain the approval of any ethics committee for a project proposing a control group of hypoxemic patients to be deprived of oxygen for a long period of time. It would also be difficult to find clinicians eager to participate in such a study.

Do we need such studies at all, even in an era of evidence-based medicine? Probably not for diseases presenting with airway obstruction such as CF or bronchopulmonary dysplasia. The effects of LTOT in COPD may also be applied to those diseases. But there remains a group of diseases with restrictive patterns of ventilatory impairment. A typical feature of these diseases is a severe decrease in arterial oxygen tension and saturation during exercise. Oxygen supplementation during exercise has already been found to relieve hypoxemia and to increase exercise capacity.

Life is motion. Perhaps we should not bother too much about prolongation of the life of our patients, which would be difficult to prove anyway. We should rather concentrate on assuring them as much mobility and enjoyment of life as possible. In other words we should work on adding life to years rather than adding years to life. For this, ambulatory oxygen is the best option.

ACKNOWLEDGMENT

My sincere thanks are due to Ms Alicja Dymont for her invaluable secretarial assistance.

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Discussion

O'Donohue: In the United States one of the most common reasons for prescribing oxygen, other than in COPD, is for patients with pulmonary fibrosis or with terminal carcinoma who become hypoxemic. Both of these are short-term. Their life expectancies are short, and we don't really expect to see extensions of life with the use of oxygen. But the important thing is the quality of life, and in none of your studies did I see that addressed. You just spoke to it, but did any of those studies look at quality of life and relief of dyspnea in patients with far advanced disease?

Zieliński: I have no data about the quality of life of non-COPD patients. Maybe Dr Wedzicha has.

Wedzicha: I don't have any data on quality of life in IPF either. I must say, though, I don't think we can ever do randomized trials with IPF patients. They get significant symptomatic benefit from oxygen therapy because they get a lot of arterial oxygen desaturation on exercise. I find they very quickly go on to 24-hour oxygen therapy. There are also some other important conditions, like CF. Several randomized control trials were done some years ago. The other group that was mentioned was terminal cancer, where, again, we are basically treating dyspnea. There was an interesting paper in *The Lancet* not long ago showing that these people do get improvement in symptom score,[1] so I really don't think we should be looking for randomized trials in these conditions. I think we really are looking more for symptomatic improvement.

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MacIntyre: You're looking for symptomatic improvement, but don't you need a controlled, randomized trial? In fact, it seems to me that in this population, where mortality is not affected, it might be a better place to do the randomized trial. The ethical issues that you described in COPD don't exist.

Wedzicha: Yes, but I don't think I could deny oxygen to a patient with severe end-stage pulmonary fibrosis who gets symptomatic improvement from oxygen. If I don't give it, their primary care physician will give it. These are people where I just don't think we can do trials.

O'Donohue: Not too long ago, in Canada, the Minister of Health was having a problem because physicians were ordering oxygen for palliation when the patients did not have hypoxemia as a cause of dyspnea. Not all dyspnea is due to hypoxemia or corrected by oxygen. So I agree with Neil. You still need to look at the "symptomatic" use of oxygen and study it.

MacIntyre: You don't have data that says they are better. This is a subjective response on the part of the patient.

Wedzicha: I certainly do not support giving oxygen to patients who are not chronically hypoxemic. I'm talking about patients with IPF who are chronically hypoxemic. The same goes for cancer patients who are chronically hypoxemic. I think I've been slightly misunderstood. But I just don't feel I can do a randomized, controlled trial on somebody who has pulmonary fibrosis and who has got chronic hypoxemia.

MacIntyre: Do we even have pre/post data though? Do we have any data other than the fact that their P_{O_2} gets better and there's this subjective impression that they look like they're better?

Wedzicha: The reason we don't have pre/post data is that patients deteriorate so quickly. They all die by 2 years. We are very short of data in these patients. There are some data around in the literature about dyspnea.

Pierson: I don't think you'd have to study these people for the remainder of their lives. One month on, one month off, in a randomized fashion might suffice to answer the kinds of questions that Walter and Neil are talking about.

Zieliński: Regarding the Zinman study, there are some objections to the study protocol. First, the studied group was very small (28 patients in both arms, treated and nontreated). Second, the patients were treated with oxygen during sleep. I think CF patients do not desaturate much during sleep. They

would need oxygen during the day when they exercise. Perhaps giving them oxygen during the daytime would be more beneficial. Although survival wasn't affected, patients treated with oxygen had more days at school and more days at work than those in the control group.

Criner: I have another view on the need for investigation of this topic. I think that subjecting it to investigation may change our prescription habits in the IPF patient group, compared to other patient groups, for a couple of reasons. First of all, what Dave (Pierson) presented earlier from a pathophysiologic standpoint, in these patients with a higher shunt fraction, their doses of supplemental oxygen may be much higher than what we usually prescribe in other patient groups. As a transplant referral center, we find that patients prescribed supplemental oxygen treatment for IPF are not appropriately dosed, or they're way undertreated with the doses prescribed. The timely correct oxygen prescription may also be more important in this patient group, because this group doesn't have time to enable adaptive mechanisms that would counterbalance hypoxemia and circumvent the development of a hypoxic state. I believe that in IPF patients these factors necessitate the prompt prescription of the correct dose of supplemental oxygen.

Long-Term Oxygen Therapy vs Long-Term Ventilatory Assistance

Jadwiga A Wedzicha MD

Introduction
Physiologic Considerations
Early Experience with Noninvasive Ventilation in Chronic Obstructive Pulmonary Disease
Positive-Pressure Techniques
Negative-Pressure Techniques
Nasal Intermittent Positive-Pressure Ventilation
Evidence for Benefit in Chronic Obstructive Pulmonary Disease
Longer-Term Outcome in Chronic Obstructive Pulmonary Disease
Recommendations for Use in Chronic Obstructive Pulmonary Disease
Bronchiectasis
Cystic Fibrosis
Summary

[Respir Care 2000;45(2):178–185] *Key words: long-term oxygen therapy, chronic obstructive pulmonary disease, noninvasive ventilation, nasal intermittent positive-pressure ventilation, bronchiectasis, cystic fibrosis, nocturnal hypoventilation.*

Introduction

Domiciliary long-term oxygen therapy (LTOT) is one of the few interventions for chronic obstructive pulmonary disease (COPD) that improves survival, when used for ≥ 15 hours a day.[1,2] LTOT has also been shown to have a number of important physiologic benefits, though it has little effect on other factors, including health-related quality of life or reduction in disease exacerbation. Many of these COPD patients exhibit marked abnormalities of overnight ventilation, with a consequent derangement of blood gases and ventilatory muscle dysfunction, which may contribute to the progressive deterioration seen in this group.[3–5] It has therefore been suggested that ventilatory support may confer further advantages when used in addition to LTOT.

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Early studies in patients with respiratory failure due to COPD, using mainly negative-pressure techniques, largely found no benefit from ventilation.[6–8] However, early uncontrolled studies of COPD patients on noninvasive positive-pressure ventilation (NPPV), mainly using nasal masks, showed that it was possible to apply ventilation to these patients, as was previously found in chest wall and neuromuscular disease. The addition of NPPV could produce significant improvements in arterial blood gases (ABGs) and control of nocturnal hypoventilation.[9–11]

Physiologic Considerations

In COPD patients, ventilation is reduced during sleep, with further reduction in ventilation during rapid-eye-movement (REM) sleep. This decrease in ventilation is largely due to reduction in tidal volume, and is not usually associated with apnea.[12] The hypoventilation will cause considerable arterial oxygen desaturation at night, which may be an important factor in the development of chronic pulmonary hypertension.[4–5] During sleep there is a reduction in carbon dioxide responsiveness[13] and an increase in upper airway resistance, leading to further respiratory insufficiency.[14] Other mechanisms of arterial oxygen desaturation include a higher physiologic dead space in COPD

patients, effects of hyperinflation, and ventilation-perfusion inequalities.

Nocturnal arterial oxygen desaturation may be corrected with supplemental oxygen therapy, though this is associated with worsening hypercapnia, especially in the early hours of the morning,[15] and little effect is observed on daytime blood gases. A study of patients with stable COPD by means of sequential polysomnography and overnight blood gas monitoring noted a relationship between the fall in arterial oxygen saturation overnight and progression of the underlying disease, particularly in those patients with concomitant hypercapnia.[16] The deterioration in hypoventilation and hypercapnia may further impair diaphragmatic function[17] and respiratory mechanics and eventually have a deleterious effect on central respiratory drive.

It has been suggested that in COPD the respiratory muscles are subject to fatigue and that improvements in lung mechanics and a consequent reduction in work load may facilitate muscle rest and relief of fatigue. Several investigators have shown that the application of noninvasive ventilation, using both positive-pressure and negative-pressure techniques, may produce significant reductions in diaphragmatic electromyogram activity and work of breathing.[7,18–20] However, no long-term controlled study has shown an improvement in measures of ventilatory muscle strength with the addition of assisted ventilation.

Evidence is emerging that the mechanism of reversal of hypoventilation and thus improvement of ABGs during the daytime is an improvement in carbon dioxide responsiveness. Hill et al showed that withdrawal of noninvasive ventilation for a period of one week in patients with chest wall and neuromuscular disease produced deterioration in symptoms and nocturnal hypoventilation, but no effects were seen on respiratory muscle function.[21] Similar results were reported by Goldstein et al in patients with neuromuscular disease, suggesting that the application of noninvasive ventilation improves the sensitivity to carbon dioxide.[22] Elliot et al, in a study of nocturnal nasal ventilation in COPD patients, showed that patients whose blood gases improved had associated increased chemosensitivity to carbon dioxide.[23] However, they were unable to demonstrate any effects on respiratory muscle fatigue with the addition of NPPV.

Patients with hypoxic COPD also have sleep disruption, though the effects of supplemental oxygen therapy on sleep quality have been variable, with some studies showing benefit and others not documenting any effect.[15,24] Although some of these patients may have coincident sleep apnea, sleep disruption in COPD is not usually caused by the effects of upper airway obstruction. COPD patients may be seen to arouse during episodes of oxygen desaturation, though sleep disruption is also present in COPD patients who do not desaturate at night.[25] Hypercapnia may be worsened overnight with the addition of supplemental oxygen therapy. An acute elevation of arterial partial pressure of carbon dioxide (P_{aCO_2}) during the night may contribute to arousals and thus impaired sleep quality.[26] Control of nocturnal hypercapnia through nasal ventilation may improve sleep quality and thus result in improved neuropsychological performance and quality of life.

Early Experience with Noninvasive Ventilation in Chronic Obstructive Pulmonary Disease

Positive-Pressure Techniques

Positive-pressure ventilation (PPV) through a tracheostomy has been widely used in France, although controlled studies of its efficacy in COPD patients are lacking. Robert et al showed that COPD patients had a worse prognosis than patients with other conditions requiring assisted ventilation, such as kyphoscoliosis or the sequelae of tuberculosis.[27] The survival in their COPD group, treated by tracheostomy, was similar to that of the British Medical Research Council long-term oxygen trial.[2] However, a further multicenter study from France of 259 COPD patients showed an improved survival, compared to that of the British Medical Research Council group for the first 4 years, and then the survival curves became similar.[28] The suggestion was made that assisted ventilation in COPD should be used at an earlier stage in the natural history of the condition to obtain increased benefit.

Negative-Pressure Techniques

Negative-pressure ventilation, although effective in patients with chest wall and neuromuscular disease, has been less successful in COPD patients, mainly due to poor patient tolerance of and compliance with the technique. Musculoskeletal and chest discomfort are common with negative-pressure devices, and the equipment tends to be more cumbersome. Upper airway obstruction may also be potentiated with the use of negative-pressure equipment.

A randomized crossover study comparing the pneumosuit negative-pressure ventilator with standard care in 20 stable COPD outpatients over 6 months showed no improvement in blood gases, maximal inspiratory pressure, maximal expiratory pressure, or exercise tolerance.[8] However, 11 of the 20 patients withdrew from the study because of inability to tolerate the ventilator. Almost all the patients were dissatisfied with the treatment and used it for 4.1 hours daily, considerably less time than recommended. Patients most frequently complained of musculoskeletal pain and inconvenience with the negative-pressure ventilation.

Another randomized study of negative-pressure ventilation in COPD using the pneumosuit device was reported in hospital patients.[6] The trial, involving 16 COPD pa-

tients, aimed to determine whether negative-pressure ventilation, when added to a standard exercise training and pulmonary rehabilitation programme, improved ventilatory muscle function, perception of well-being, and exercise performance. There was no physiologic advantage documented from the use of negative-pressure ventilation and, again, compliance with the equipment was poor. However, as in the previous study described, the COPD patients had only modest hypercapnia.

Shapiro et al reported a study of 184 COPD patients who were randomized to receive either active negative-pressure ventilation or sham treatment over a 12-week period of home use.[7] There were no significant changes in the active compared to the sham group with respect to the outcome measures, which included cycle exercise endurance time, severity of dyspnea, quality of life, arterial blood gas tensions, and respiratory muscle strength. The authors concluded that negative-pressure ventilation was ineffective as a method for resting respiratory muscles in patients with stable COPD. However, again, compliance with the technique was poor, with 63 patients either not using the respirators at all or stopping before completing the 12-week study period. Although the aim of the study was for the patients to use the ventilation overnight, this was unsuccessful and most of the patients were only treated during the daytime, with the vast majority receiving < 3.5 hours of ventilation daily. The aim of the study was to obtain at least a 50% reduction in the amplitude of the diaphragmatic electromyogram signal, with an average ventilator use of 5 hours daily, but this objective was only achieved in 29% of the patients.

Other studies in COPD patients have been reported using negative-pressure ventilation on an intermittent basis in hospital. One study showed improvement in blood gases, though the design was uncontrolled, with small numbers of patients.[29] However, in this study, the patients were significantly hypercapnic. Another controlled study suggested a beneficial effect on ventilatory muscle function in those patients who received negative-pressure ventilation for 3 to 6 hours daily for 3 consecutive days, though no long-term follow-up or therapy with negative-pressure was available.[30]

Nasal Intermittent Positive-Pressure Ventilation

Evidence for Benefit in Chronic Obstructive Pulmonary Disease

Following the success of NPPV in correcting hypoventilation and daytime blood gases in chest wall and neuromuscular disease, treatment was extended to COPD patients. Early uncontrolled studies showed that ABGs improved in patients treated with NPPV, with improvement in sleep quality.[9,10,31] Results were more favorable than those achieved previously with negative-pressure ventilation. However, in these studies compliance with ventilation was not as good as in patients with restrictive chest disease. The patients who showed benefit were those who also had significant daytime hypercapnia and in whom nocturnal hypercapnia could be successfully reduced by overnight ventilation.[10] Supplemental oxygen was not used routinely with the NPPV in some early studies. This may have reduced benefit because overnight oxygen saturation levels were not adequately corrected with NPPV alone.[18]

Strumpf et al performed a randomized crossover study of nasal ventilation using a bi-level positive airway pressure ventilator (BiPAP, Respironics, Pittsburgh, Pennsylvania) in 19 COPD patients.[32] Compliance proved to be a major problem in the study, and only 7 patients completed both arms of the protocol, the poor compliance mainly resulting from problems with the nasal mask interface. This study showed no changes in pulmonary function, respiratory muscle strength, gas exchange, exercise endurance, sleep efficiency, sleep quality, oxygenation, or dyspnea ratings between the 2 arms of the trial. The only improvements observed were in neuropsychological function. Although these patients had severe air flow obstruction, they showed less severe derangement of blood gases and, in particular, lesser degrees of hypercapnia, with some patients in the normocapnic range.

Meecham Jones et al reported a randomized crossover study of nasal pressure support ventilation plus oxygen therapy, compared to domiciliary oxygen therapy alone, in 18 hypercapnic COPD patients.[11] The aim of this study was to investigate the effect of the addition of nasal intermittent positive-pressure ventilation (NIPPV) to patients already established on LTOT. Following a run-in period, each patient received randomly NIPPV plus oxygen and oxygen alone for 3-month periods. There were significant improvements in daytime ABGs, with the mean arterial partial pressure of oxygen (P_{aO_2}) rising from 44.3 mm Hg after the oxygen period to 50.2 mm Hg after the addition of NPPV, and P_{aCO_2} falling from 57.0 mm Hg to 52.5 mm Hg (Fig. 1). After the addition of NPPV there was also improvement in overnight P_{aCO_2}, total sleep time, and sleep efficiency, suggesting that control of hypoventilation in these patients is effective and leads to improved sleep quality. The improvement in daytime blood gas values was correlated with the change in overnight P_{aCO_2} (Fig. 2). This suggests that the patients who showed the greatest improvement in P_{aCO_2} with nocturnal nasal ventilation are likely to gain the greatest benefit from the treatment.

Compliance in this study was much better than most previous studies of noninvasive ventilation in COPD, with 14 of the 18 patients completing the study, and only 1 patient was withdrawn because of inability to tolerate the equipment. Previous studies had mainly initiated NPPV in outpatients,[32] whereas in this study all patients had nasal

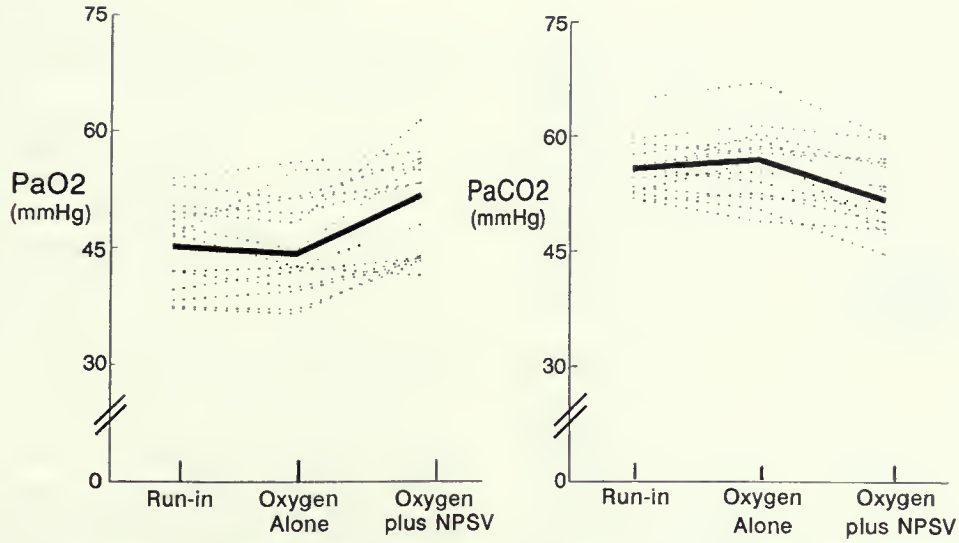

Fig. 1. Changes in daytime arterial blood gases (arterial partial pressure of oxygen [P_{aO_2}] and arterial partial pressure of carbon dioxide [P_{aCO_2}]) during oxygen therapy alone and then with the addition of nasal pressure support ventilation (NPSV) for a 3-month period. Individual data are shown for 14 COPD patients, with the mean values as solid lines. (From Reference 11, with permission.)

ventilation initiated as inpatients, allowing them maximum support and education while they became familiar with the equipment. Improved compliance may also have been due to the greater degree of hypercapnia in these patients and consequent benefit achieved with ventilation.

As progressive COPD is associated with significant disturbance of the activities of daily living, measures of quality of life are essential for assessing the effectiveness of therapies.[33] Studies of LTOT have generally not demonstrated any benefit with respect to quality of life.[34] Meecham Jones et al measured quality of life using the St George's Respiratory Questionnaire (SGRQ), which provides a disease-specific measure of quality of life, with 3 component scores (symptoms, activities, and impacts) and a total score.[35] The combination of NIPPV with oxygen therapy produced significant improvements in symptom, impact, and total quality of life scores, as compared to oxygen alone. There was no change in the activity component in the 2 study groups and no change in 6-minute walking distance after ventilation. A study involving patients with restrictive ventilatory disorders showed that quality of life was related to sleep quality.[36] Although these findings were not confirmed in the study by Meecham Jones et al, it is possible that improvements in sleep time and efficiency contribute to parallel improvements in health-related quality of life. Seemungal et al recently found that quality of life assessed using the SGRQ is strongly related to exacerbation frequency.[37] It is possible that the observed improvement in health status reflects reduction in exacerbation frequency, though this was not addressed in this particular study. NPPV may also protect the patient against deterioration in blood gases during an exacerbation, and this may be the mechanism of any observed survival benefit.

Perrin et al followed 14 patients with hypercapnic COPD over a 6-month period and found similar improvements in daytime ABGs.[38] This study is also of interest in that improvements were also found in the total SGRQ score and in the impacts component of the SGRQ score.

Another small study that investigated the effects of the addition of NPPV to oxygen therapy in severe COPD found no significant benefit from NPPV after only 2 weeks of therapy.[39] However, acclimatization to the ventilator may take longer than 2 weeks, so the study time was too short. In that study, patients had problems with higher levels of inspiratory positive airway pressure ($\geq$ 15 cm H_2O) and this suggests that they required a longer period of acclimatization to the ventilator.

Longer-Term Outcome in Chronic Obstructive Pulmonary Disease

There are some data on longer-term outcome with NPPV in COPD, though these data are mainly from uncontrolled studies. Leger et al followed 50 COPD patients on NPPV and observed a significant decrease in P_{aCO_2} in long-term survivors over 2 years, again emphasizing the importance of NPPV for control of hypoventilation and outcome.[40] Probability of continuing nasal ventilation was lower in COPD patients, at 56%, compared to patients with kyphoscoliosis, at 80%, and sequelae of tuberculosis, at 76%. In a United Kingdom series of 33 COPD patients treated with NPPV over a 5-year period, the probability of continuing ventilation was lower, at 43%,[41] which is comparable to

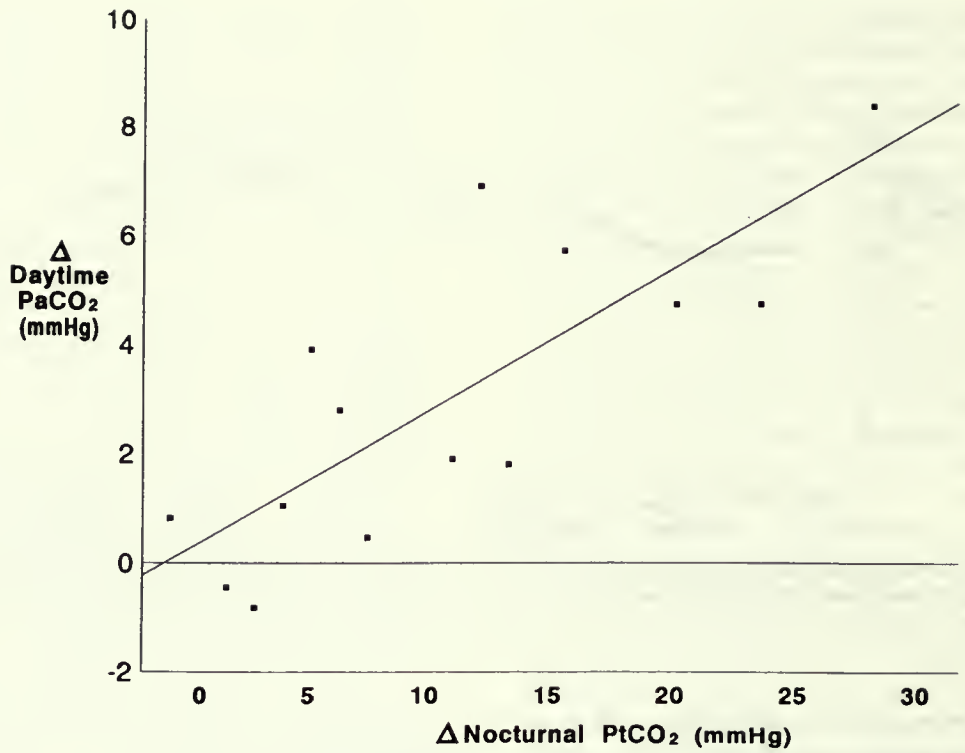

Fig. 2. Relationship between the change in mean daytime arterial partial pressure of carbon dioxide (P_{aCO_2}) and change in mean nocturnal partial pressure of carbon dioxide measured transcutaneously (P_{tCO_2}) after 3 months of nasal ventilation in 14 COPD patients. The degree of improvement in daytime P_{aCO_2} was correlated with the improvement in mean overnight P_{tCO_2}, with those patients showing the greatest improvement in nocturnal P_{tCO_2} having the largest changes in daytime P_{aCO_2}. (From Reference 11, with permission.)

survival achieved with LTOT.[1,2] However, patients included in this series were at the severe end of the spectrum of chronic respiratory failure, and most were deteriorating despite LTOT. It is likely that NPPV was instituted too late in the natural history of the condition to achieve optimum benefit.

A study of NPPV in COPD patients who were unable to tolerate LTOT showed that survival was similar to those able to take oxygen therapy and better than historical controls.[42] Factors predicting a poor survival included high overnight transcutaneously-measured carbon dioxide tension, again emphasising that control of nocturnal hypoventilation is an important factor in the use of NPPV in hypercapnic COPD.

Another recent small uncontrolled study of NPPV in COPD found that hospital admissions and primary care consultations were halved with NPPV, though this type of analysis is subject to bias, because the use of NPPV involves more supervision and hospital visits.[43]

A recent study divided hypercapnic COPD patients into two groups, depending on whether the patient could tolerate NPPV. Although there was no difference in mortality between the two groups, patients taking NPPV had a reduction in intensive care admissions, compared to those on LTOT alone.[44] However, this study has some important limitations in that the patients were not randomized and were only selected on ability to tolerate NPPV. The results of randomized controlled trials comparing long-term outcome with NIPPV compared to oxygen therapy are awaited.

Recommendations for Use in Chronic Obstructive Pulmonary Disease

Results from randomized studies described above suggest that there is now sufficient evidence for the use of NPPV in selected COPD patients (Table 1).[11] However, there is no evidence from controlled trials that NPPV improves survival in hypercapnic COPD. Patients must have daytime hypercapnia and documented nocturnal hypoventilation that can be controlled with the chosen ventilator. Patients with the most effective reduction of hypercapnia during the night will be expected to achieve the most benefit from the addition of NPPV.[11] Thus, prior to establishing a patient on home NPPV, investigation with overnight monitoring of hypoventilation and the response to NPPV is required. COPD patients with hypercapnia and considerable sleep disruption will be particularly suitable for long-term ventilation, because sleep quality has been shown to improve with NPPV. Obstructive sleep apnea may coexist with COPD and must be assessed prior to prescribing home NPPV.

Table 1. NPPV in COPD—Selection Criteria of Patients for Long-Term Oxygen Therapy

Daytime hypercapnia
Nocturnal hypoventilation
Established on long-term oxygen therapy
NPPV improves control of hypoventilation
Poor sleep quality with frequent arousals
Exclude end-stage disease—stability of blood gases
Appropriate support and education for patient
Adequate patient motivation

NPPV = noninvasive positive-pressure ventilation; COPD = chronic obstructive pulmonary disease.

Initiation of NPPV for COPD on an inpatient basis may improve compliance,[10,11] though this will increase treatment costs. Compliance is frequently affected by complications related to the nasal mask used for ventilation. Although nasal mask complications are common at the start of ventilation, most resolve with specific attention.[45] Early identification of nasal mask complications and intervention will improve compliance and thus efficiency of NPPV. Patients chosen for home NPPV must be appropriately motivated and prepared to accept the ventilator at home. A ventilation acclimatization period may allow careful adjustments of the settings to increase patient comfort. In some COPD patients the presence of intrinsic positive end-expiratory pressure may produce asynchrony between the ventilator and the patient's respiratory efforts.[46] Care must be taken during the initiation of NPPV to ensure adequate capture of the ventilation. Patients will be monitored with blood gases and adjustments of the ventilator may be later required, especially when the disease eventually progresses. Various types of ventilators are available for NPPV in COPD, and the choice will depend on local experience, cost, and patient acceptance. Both volume-cycled and pressure-cycled ventilators are effective in patients with hypercapnic COPD.[47] Equipment for pressure support ventilation is generally less expensive than that required for assist control ventilation, and has been used in a number of reported studies.

The point in the natural history of hypercapnic COPD at which NPPV treatment should commence has not been evaluated. Although LTOT improves survival in patients with hypoxemic COPD, eventually patients die with progressive irreversible air flow obstruction, with blood gas measurements deteriorating before death.[48] Studies that found benefit from NPPV in COPD recruited mainly stable patients. However, patients are often referred for further management when blood gas measurements are deteriorating, despite optimum management and LTOT. Meecham Jones et al reported a study of 9 patients with advanced hypercapnic COPD and hypoxemia refractory to oxygen therapy (P_{aO_2} < 60 mm Hg on supplemental oxygen).[49] The addition of long-term NPPV to LTOT in these patients produced no improvement in clinical condition or blood gases. This suggests that chronic NPPV should be used at an earlier stage in the natural history of the condition, before rapid clinical deterioration occurs. However, several studies have found that patients with acute respiratory failure due to COPD can be successfully treated with NPPV.[50–52] Further studies are required to assess the optimum time for starting long-term NPPV in COPD patients.

Bronchiectasis

Patients with bronchiectasis may be difficult to distinguish in clinical practice from COPD patients. Pulmonary function tests will show an obstructive defect, with or without a restrictive pattern. With disease progression ventilation-perfusion ratio worsens and hypoxemia develops. Later the patient will develop hypoventilation with hypercapnia, though at this stage there is often severe end-stage lung disease. In addition to the progressive lung disease, patients with bronchiectasis have copious sputum production and recurrent infection, which cause further problems during assisted ventilation and weaning.[53]

In the French series on patients using PPV through a tracheostomy, survival was inferior for those with bronchiectasis, compared to patients with COPD and chest wall deformity or neuromuscular disease.[27] In a recent report from the United Kingdom, in a series of patients with bronchiectasis and respiratory failure treated by NPPV, the major outcome measure was probability of continuing NPPV, which was < 20% after two years.[41] The NPPV was started late in the natural history of the condition and the patients became increasingly ventilator dependent during the treatment period. All the patients with bronchiectasis had been referred for consideration of heart–lung transplantation. In a study by Leger et al,[40] 25 patients were treated with NPPV for bronchiectasis. There was a higher probability of continuing NPPV than in the British study. However, significant change in blood gas measurements was not seen and, in contrast to the other indications for NPPV, there was no reduction in days of hospitalization for respiratory illness. One retrospective study of NPPV using volume-cycled ventilators in bronchiectasis found that, although the P_{aCO_2} was not reduced, it was stabilized with NPPV, and the number of days in hospital was reduced.[54]

Cystic Fibrosis

Intubated cystic fibrosis patients may have a prolonged clinical course in the intensive care unit and considerable difficulties with weaning. The availability of heart–lung transplantation provides hope to end-stage patients with cystic fibrosis and progressive respiratory failure, because

such patients may be removed from transplantation lists if intubated. NPPV has been used in the management of these patients in an effort to avoid complications of intubation and to serve as a bridge to transplantation.[55]

Piper et al treated 4 cystic fibrosis patients who had hypercapnic respiratory failure for up to 18 months.[56] Within a few days of starting NPPV, hypercapnia was reduced and patients reported improved length and quality of sleep. After stabilization, all the patients were discharged home, with improvements maintained for up to 18 months. Hill et al followed 10 patients with cystic fibrosis awaiting transplantation who were treated with home NPPV for up to 15 months.[57] At 3 months after treatment they observed improvements in forced vital capacity, P_{aCO_2}, and a significant reduction in the number of hospital inpatient days. However, in this study patients were generally started on NPPV late in the natural history of their disease. Earlier use may have advantages and requires study.

Summary

The use of positive-pressure nasal ventilation in combination with LTOT in stable COPD patients with hypercapnic respiratory failure controls hypoventilation and improves daytime ABGs, sleep, and quality of life. Nasal ventilation in COPD is unlikely to produce benefit unless used with supplemental oxygen therapy at night. The patients who show the greatest reduction in overnight P_{aCO_2} with ventilation are the patients most likely to benefit from long-term ventilatory support. Although there is now evidence for short-term benefit from NPPV in hypercapnic COPD, large multicenter studies with survival, exacerbations, and hospital admissions as the primary end points are required to evaluate longer-term effects of this potentially important intervention.

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Discussion

MacIntyre: In the United States there's quite a controversy about NPPV in COPD. Walter and I and maybe some other people in this room have been involved with the United States Medicare system on reimbursement strategies for this particular modality. One of the key points of contention in the United States is the issue of a backup rate. The reason that's important in the United States and probably not so important in the rest of the world is that if you supply an NPPV device with a backup rate the reimbursement rate is substantially higher than for a system without a backup rate, so there's a huge financial difference, and the issue is whether COPD patients ought to have backup rates. The way it's falling out in Washington, DC, right now is that COPD patients are likely not to be reimbursed for the much more expensive backup rate system unless there is a sleep study showing that the backup rate is important. So I was intrigued with your comment that in the Meecham Jones study backup rates were not used,[1] but in spite of that you have taken the position that they ought to have backup rates. The manufacturers in the United States would absolutely love you. The Medicare folks are going to be very upset with you. I'm curious why you chose to use the backup rate.

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Wedzicha: We have no studies on this point. We have got one study in which we compared no backup to backup. This was in a combination of COPD patients and chest wall disease, and we found not much difference between the two rates. That's how we justified using no backup rate in these studies. We have started using backup rates on some of the patients who have more severe nocturnal hyperventilation. But we've never done any more detailed work on that point.

Petty: As Priestley was a heretic, I guess I am too, because I don't think that carbon dioxide is the enemy. I think that work of breathing is the enemy. I will respectfully say this: I never do what you do. What I do is try to encourage chronic compensated carbon dioxide retention, including use of narcotics to blunt respiratory drive, so that the P_{CO_2} will rise beyond that magic threshold of about 80, at which point, if appropriate bicarbonate is generated by the kidneys, there's immense relief of dyspnea. It's as if the respiratory center says "To hell with it, I'm not going to work so hard to breathe any more." And as we published years ago, many patients live at home quite comfortably with a P_{CO_2} of 100, compensated, breathing at a minute ventilation of 2–3 L/min.[1] They may get very sleepy for a while as they get to end-stage disease; they may sleep for 2 or 3 days, then they wake up. In this so-called sleep therapy all of the endorphins come out of the brain. So I think it's a mistake to drive P_{CO_2} down. I think we should encourage P_{CO_2} to go high so homeostasis at high P_{CO_2} can relieve dyspnea.

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Wedzicha: Tom, the problem with patients with P_{CO_2} of 100 is that they're very vulnerable to exacerbation. And these are the people who come backwards and forwards into hospital. I must say that when we treat these people one of the first outcomes is that we see them less at hospital admissions. If we do see them, their admissions are shorter.

Benditt: You brought up the question of using a sham ventilator. One thing that always impressed me about that Shapiro and Macklem study[1] was the fact that their sham group showed improvement too, indicating that the equipment had some placebo effect. What do you think about that, and do you think that is an issue?

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Wedzicha: I think you will always get placebo effects from the equipment; certainly you do with sham continuous positive airway pressure. I think that's why we need randomized controlled trials. However, with a ventilator I think that, if there is a placebo effect, it probably will be relatively small. The other problem is practicality. You're going to have to give a very low pressure to these patients. You're effectively going to give them continuous positive airway pressure. I can't see any other way of doing a sham procedure. And I have some problems if you do a two- to three-year study. The continuous positive airway pressure sham procedures were done for three months. That's totally different than doing it for a two- to three-year period. If you want to catch exacerbations, you've got to study for a few years.

Kacmarek: If I understood your conclusions correctly, you are recommending the use of noninvasive ventilation on a long-term basis in selected patients. However, I must admit I was not convinced that you've presented any information that would convince me to do long-term noninvasive ventilation with COPD patients. We clearly have a problem with compliance. Even in the hospital when noninvasive ventilation is used short-term for acute exacerbations, most patients demand discontinuation as soon as they recover from the exacerbation. It's difficult to get this group to tolerate NPPV in the hospital, let alone at home. The information you presented about reduction in exacerba-

tions and changes in quality-of-life scoring is where I have concerns. Did these data separate patients who were on noninvasive ventilation from patients who weren't, or simply separate patients who had frequent exacerbations from those with nonfrequent exacerbations? My understanding is that there are no randomized trials data to show that noninvasive ventilation does anything except in selected patients, where you see some improvement in overall gas exchange.

Pierson: You're talking about COPD? Not neuromuscular disease?

Kacmarek: COPD only.

Wedzicha: In COPD, as I said, we have no randomized controlled trial evidence that exacerbations are reduced. The data I showed was from a population of patients with severe COPD. We desperately need a long-term trial with outcome measures very carefully defined. My prejudice is that survival will not be improved. The forced expiratory volume in the first second is a strong predictor of mortality, but there is, I think, increasing uncontrolled data that hospital admissions improve. Hospital admissions is a difficult outcome measure because it is dependent, certainly where I work, on a lot of other factors. But I think we now know how to monitor patients much more carefully, and I think that's the line we have to follow.

Pierson: There's a tendency to treat LTOT versus nothing, and NPPV versus nothing, as if it were a simple black-and-white matter, like putting someone on isonicotinic acid hydrazide for a tuberculosis skin test conversion, whereas I think NPPV especially is a lot more like putting someone on Coumadin to maintain anticoagulation. If you've ever managed someone long-term on Coumadin, it drives you crazy trying to keep them in a therapeutic range, because it takes so much titrating and adjustment. It's hard for me to keep track of the different techniques and monitors that have been used to gauge whether the ventilation is actually unloading the muscles, and I wonder if you have a comment about how you adjust the machine and the targets.

Wedzicha: I think one other point I meant to answer to Bob on this is that, if you are, for instance, doing trials, you have to carefully control the control group because you're going to give one group very much more attention. I think these people do need a lot of attention. We base our initial ventilation settings on daytime gases, using ear lobe blood gases. The patients have an overnight sleep study when we titrate the ventilation to get optimal improvement in overnight P_{CO_2}. And then when we see the patients three-monthly, we check the ear lobe gases and adjust the settings. We have also found that where we started with pressure-cycle ventilation we have had to change to volume-cycle ventilation in some of these patients, especially if there is a lot of airway disease.

O'Donohue: There was an NAMDRC (National Association for Direction of Respiratory Care) consensus conference regarding NPPV, and most of the professional organizations represented at this conference were there. The results of that conference are published in *Chest*.[1]

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Pierson: Was this consensus conference on NPPV in COPD?

O'Donohue: No, but COPD was a major component of the conference.

Pierson: Can you tell us anything about their conclusions with respect to this particular topic? Neil?

MacIntyre: Their recommendations in COPD actually go along pretty much with what you said. P_{CO_2} above 55 was the threshold, and that recommendation was adopted by the United States Health Care Finance Administration. There was a proviso for P_{CO_2} between 50 and 54, but they had to have accompanying symptoms of sleep disorders, morning headaches, or dyspnea that didn't respond to oxygen, and a few other things. The federal government at the moment is not accepting that recommendation for P_{CO_2} values between 52 and 54. On the issue of the backup rate, we couldn't come to consensus, and we left it at physician discretion. As I mentioned during my comments, the federal government did not want to leave it to physician discretion. They said the default condition was going to be no backup rate, the cheaper system, and that a polysomnogram or some other evidence that they needed a backup rate must be provided to get the more expensive system. Interestingly enough, in terms of whether to continue it, it was decided that you ought to give patients a two-month trial, and if at the end of the two months they were (a) still using it, and (b) said they liked it, then they could keep it. If they didn't meet those two requirements, the machine would be yanked from the home. Pat Dunne and I talked about that, and the psychological issues of pulling the machine out of the home, even if it's not being used, may turn out to be quite problematic.

Wedzicha: In the United Kingdom there are 18,000 people on LTOT and 12,000 have COPD, so our projection is that there are about 6,000–7,000 patients who could potentially benefit. Does anyone know the equivalent United States figure?

Petty: I think there are about a million people on home oxygen, of which 25% might have hypercarbia, so 250,000 would be my guess.

Hypoxemia During Sleep in Patients with Chronic Obstructive Pulmonary Disease: Significance, Detection, and Effects of Therapy

Walter J O'Donohue Jr MD and Teri J Bowman MD

Introduction
Mechanisms of Oxyhemoglobin Desaturation During Sleep
Significance of Nocturnal Oxygen Desaturation in Chronic Obstructive Pulmonary Disease Patients
Recommendations for Diagnosis and Therapy in Patients with Nocturnal Oxygen Desaturation
Summary

[Respir Care 2000;45(2):188–191] *Key words: nocturnal hypoxemia, nocturnal oxyhemoglobin desaturation, oxygen therapy, chronic obstructive pulmonary disease.*

Introduction

Nocturnal oxyhemoglobin desaturation (NOD) occurs transiently in many normal individuals, and is often more frequent, more severe, and of longer duration in patients with chronic obstructive pulmonary disease (COPD).[1,2] The significance of NOD in COPD patients who have mild or no hypoxemia while awake is a subject of considerable controversy, and is important in regard to the possible effects of therapy on quality of life and long-term survival. In addition, if nocturnal oxygen therapy were prescribed for every COPD patient who has transient episodes of oxyhemoglobin desaturation during sleep, a substantial increase in health care costs would result, with no definitive evidence of medical benefit.[3]

Multicenter clinical trials in both North America and the United Kingdom have demonstrated the value of continuous or nearly continuous oxygen in COPD patients who are hypoxemic while awake.[4,5] In these patients the continuation of oxygen therapy during sleep appears to be critical to both survival and the improved health effects observed. In the Nocturnal Oxygen Therapy Trial (NOTT),[4] the flow of oxygen at night was automatically increased by one liter over the flow at rest during the day, so as to avoid NOD, though nocturnal oxyhemoglobin saturation measurements to document the effect of this flow increase were not part of the study. Whether the increase in nocturnal oxygen flow had any significant effect on the final outcome is unknown. The control group of patients was receiving nocturnal oxygen only and continued to experience hypoxemia throughout the day, with adverse long-term clinical consequences.

Mechanisms of Oxyhemoglobin Desaturation During Sleep

To understand the mechanisms of sleep desaturation in COPD patients, it is first necessary to understand respiratory patterns during normal sleep. In brief, during all stages of sleep normal healthy volunteers characteristically have a lower minute ventilation than while awake because of a lower tidal volume (V_T).[6] During rapid-eye-movement (REM) sleep (the dream state) there is a further reduction in V_T, that exceeds any other stage of sleep, and V_T may be reduced by up to 73% of the awake V_T. Overall, the functional residual capacity is reduced, with a resultant maldistribution of ventilation.[7] Consequent to the reduction in V_T, normal subjects also have a 1–2% drop in arterial oxygen saturation (S_{aO_2}), with an approximate reduction in arterial partial pressure of oxygen (P_{aO_2}) from 96.5 mm Hg to 82.5 mm Hg at or near sea level.[8] The relatively small reduction in oxygen saturation in normal subjects occurs because they are higher on the oxyhemoglobin saturation curve than COPD patients.

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During REM sleep, there is generalized skeletal muscle hypotonia except for functional diaphragm and ocular movements during the phasic portion of REM sleep. The hyperinflated COPD patient tends to use the upper chest wall skeletal muscles to assist in maintaining adequate ventilation because of a flattened diaphragm. Consequently, during the hypotonia of REM sleep there is a 14% reduction in minute ventilation during REM sleep associated with phasic eye movements, which can lead to nocturnal hypoxemia.[9,10] The sleeping COPD patient entering the phasic REM period will therefore experience a combination of intercostal muscle hypotonia and an ineffective, flattened diaphragm that leads to a much more profound loss of functional residual capacity than in normal subjects. Since the thoracic gas volume of hyperinflated lungs is only slightly higher than closing volume, the functional residual capacity then drops below the closing volume of many alveoli, resulting in further reduction in the ventilation-perfusion ratio and probable microatelectasis. This is more likely to occur in the COPD patient, whose arterial oxygen saturation is already at the steep slope of the oxyhemoglobin saturation curve, with a much greater fall in S_{aO_2} than in a normal subject. In a hypercapnic COPD patient, this saturation drop occurs more rapidly because of the elevated carbon dioxide gas tension. Further pathophysiologic changes that add to the desaturation of the sleeping COPD patient are increased ventilatory dead space due to V_T reduction, increased airway resistance, impaired mucociliary clearance, blunted cough reflex, and depressed ventilatory drive.[10,11]

Significance of Nocturnal Oxygen Desaturation in Chronic Obstructive Pulmonary Disease Patients

Wynne et al[12] provided an early detailed description of sleep NOD, which was divided into two types: (1) short episodes (a few seconds to several minutes) associated with alterations in breathing, such as hypopneas and apneas, and (2) longer periods of desaturation (several minutes to > 30 min) with no specific alterations in breathing pattern. These longer episodes usually occur during REM sleep, are associated with elevations in pulmonary artery pressure (P_{pa}), and are characteristic of NOD in COPD. Figure 1 shows the effect of nocturnal oxygen therapy on nocturnal desaturation during REM sleep in a COPD patient.[13] Figure 2 shows the effect of nocturnal oxygen therapy on P_{pa} in COPD patients with NOD over a 3-year period.[14]

Since transient episodes of oxyhemoglobin desaturation are common during sleep, a definition of clinically important NOD has remained elusive. Levi-Valensi et al,[15] in Europe, defined NOD as 30% of total sleep time with S_{aO_2} at ≤ 90%. Fletcher et al,[16] in the United States, favored a definition of S_{aO_2} at < 90% for ≥ 5 minutes, with a nadir of 85% or less during REM sleep. Using these definitions for NOD, both groups found that 25–45% of patients with symptomatic COPD who are not hypoxemic while awake (P_{aO_2} > 60 mm Hg) experience NOD during sleep. Pulmonary artery catheterization measurements of these patients found pulmonary hypertension in up to 90%, even though there were no specific signs or symptoms to accompany this physiologic finding.[17,18] Mean P_{pa} continued to increase when the subjects went untreated, but was reduced by administration of nocturnal oxygen. In a 3-year retrospective multicenter study, untreated subjects appeared to have an increased risk of early death,[19] but a small prospective study to define the possible benefits of nocturnal oxygen therapy in similar subjects showed only a modest reduction in P_{pa}, with no change in pulmonary vascular resistance, survival, or clinical course.[14] In a study of 31 COPD patients with awake P_{aO_2} of 60 mm Hg and no evidence of NOD, re-evaluation after 42 months found that 8 (26%) had subsequently developed NOD.[20]

It is estimated that 14–16 million people in the United States have COPD. If 25% of the smaller number (14 million) were found to have NOD by nocturnal oxyhemoglobin saturation studies, this would represent 3.5 million patients who could qualify for nocturnal oxygen therapy. Based on previously published Medicare cost studies for home oxygen therapy,[21] the additional cost would be more than $7 billion per year, which is more than 6 times the entire budget of the National Heart, Lung, and Blood Institute.[3]

Recommendations for Diagnosis and Therapy in Patients with Nocturnal Oxygen Desaturation

Any patient who has hypoxemia with a $P_{aO_2} \leq 55$ mm Hg or $S_{aO_2} \leq 88\%$ while awake is a candidate for continuous oxygen therapy, including nocturnal oxygen. The finding of NOD without daytime hypoxemia should never be used as a criterion to justify continuous (24 h/d) oxygen therapy. If a patient is clinically unstable at the time continuous oxygen therapy is begun (such as those being discharged from the hospital while recovering from an acute exacerbation of respiratory disease), arterial blood gases should be rechecked in 1–3 months, when the patient is stable and receiving optimal medical management.[22] If hypoxemia ($P_{aO_2} \leq 55$ mm Hg or $S_{aO_2} \leq 88\%$) is still present, then continuous long-term oxygen therapy is justified indefinitely. An arterial blood gas measurement obtained with the patient awake and at rest is desirable to document the need for continuous long-term oxygen therapy. Medicare regulations also allow home oxygen therapy if the P_{aO_2} is < 60 mm Hg or the $S_{aO_2} < 90\%$ and there is evidence of cor pulmonale, right heart failure, or erythrocytosis with hematocrit ≥ 56%.

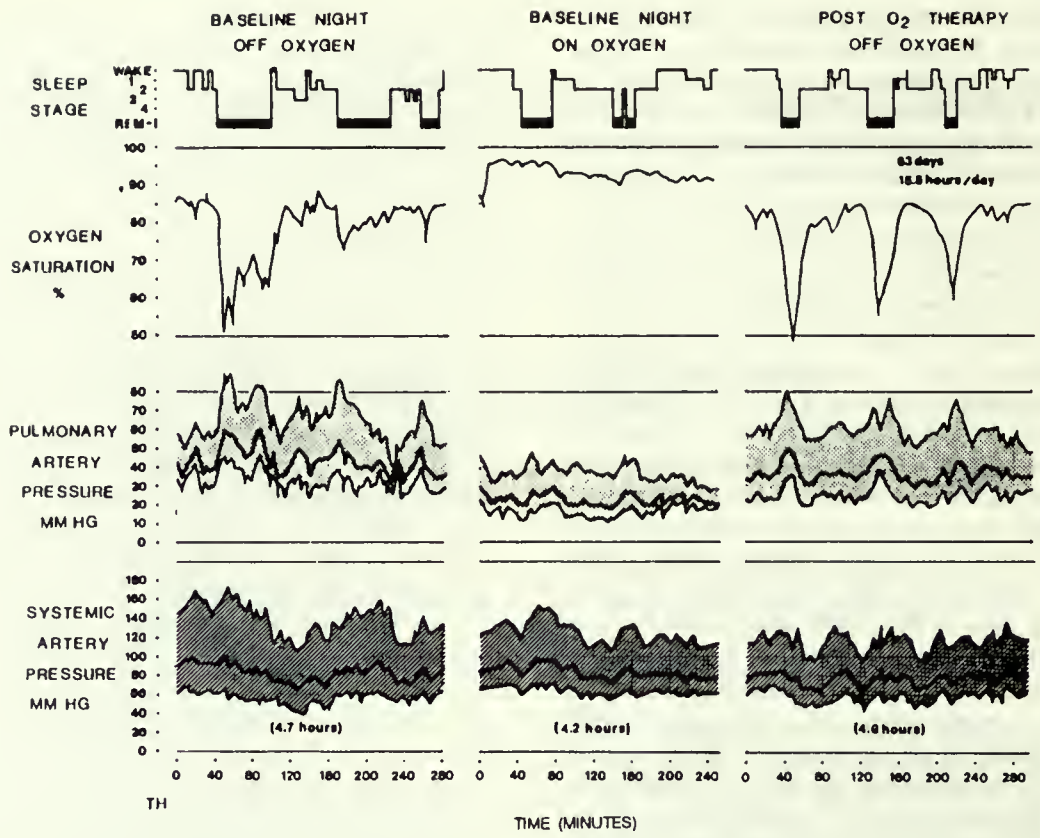

Fig. 1. Nocturnal oxyhemoglobin desaturation (NOD) in a chronic obstructive pulmonary disease patient who was not hypoxemic while awake. The column on the left shows NOD occurring during REM sleep and associated with increased pulmonary artery pressure and systemic hypertension. The middle column shows improvement with nocturnal oxygen therapy (4 L/min via nasal cannula) for 9 weeks. The right column shows findings similar to the left column when nocturnal oxygen is discontinued. (From Reference 13, with permission.)

Some patients who are not hypoxemic during the day may have clinical signs or symptoms that suggest nocturnal desaturation, such as cor pulmonale, right heart failure, or erythrocytosis. In these patients sleep studies should be performed to document NOD and to rule out other sleep disturbances such as obstructive or central sleep apnea. Appropriate therapy is then prescribed in accordance with the specific diagnosis. Medicare regulations allow reimbursement for nocturnal oxygen therapy if the P_{aO_2} is $\leq$ 55 mm Hg or the $S_{aO_2} \leq 88\%$, without specific regard for the duration of NOD. Nocturnal oxygen is also reimbursed if there is a drop in P_{aO_2} of $>$ 10 mm Hg or in S_{aO_2} of $> 5\%$ with signs or symptoms of hypoxemia, which Medicare defines as "cognitive process, restless, or insomnia."

Considering the relative ease with which nocturnal oxygen can be prescribed and reimbursed and the possibility for therapy in large numbers of patients in whom there is no established medical benefit, there is a substantial potential for misuse and abuse. Without appropriate nocturnal studies, oxygen therapy may have little benefit in patients with transient sleep desaturation and the potential for worsening disorders such as obstructive or central sleep apnea, hypoventilation syndromes, or neuromuscular respiratory failure. In many of these conditions, airway pressure devices or ventilatory assistance are the appropriate primary modes of therapy and supplemental oxygen may or may not be required. In patients with COPD and sleep hypoxemia that is correctable by supplemental oxygen alone, it is recommended that criteria such as those described by Levi-Valensi or Fletcher be utilized to document clinically important NOD.[15,16] In addition, there should exist some evidence of hypoxic tissue effect such as pulmonary hypertension, cor pulmonale, right heart failure, or erythrocytosis. Nocturnal oxyhemoglobin desaturation of lesser degrees and without evidence of organ dysfunction has not been demonstrated to have adverse health consequences, and oxygen therapy has not been shown to have medical benefit or to be cost-effective. Since pulmonary hypertension and cor pulmonale may result from NOD and may have an adverse effect on both mortality and morbidity, and since studies to date have not shown conclusive evidence of benefit from treatment of NOD in COPD patients who are not hypoxemic while awake, controlled studies are still needed and offer an excellent opportunity for future multicenter prospective investigation.

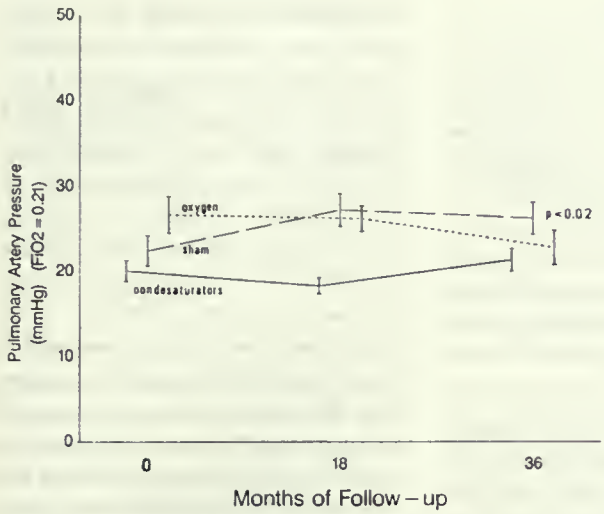

Fig. 2. Evolution of supine mean pulmonary artery pressure at 0, 18, and 36 months in 51 patients with moderate to severe chronic obstructive pulmonary disease who did not have hypoxemia while awake. The solid line represents patients (n = 13) without nocturnal oxyhemoglobin desaturation (nondesaturators) who were untreated (controls). The dotted line represents patients (n = 19) with nocturnal oxyhemoglobin desaturation who were treated with nocturnal supplemental oxygen and had a 3.7 mm Hg reduction in pulmonary artery pressure. The dashed line represents patients (n = 19) with NOD who received nocturnal compressed air and had a 3.9 mm Hg increase in pulmonary artery pressure. The difference in pulmonary artery pressure between the sham-treated and oxygen-treated groups is significant, at $p < 0.02$. F_{IO_2} = fraction of inspired oxygen. (From Reference 14, with permission.)

Summary

NOD is a common event in patients with symptomatic COPD who are not hypoxemic while awake. Up to 45% of these patients may have significant oxyhemoglobin desaturation during sleep, and most have evidence of pulmonary arterial hypertension. Although intuitively it would seem that supplemental oxygen during sleep should be of medical benefit in COPD patients with NOD, studies to date have not substantiated this idea. Medicare requirements for prescribing nocturnal oxygen are relatively liberal and there is the possibility of misuse, which would cause a substantial increase in the cost of home health care. A well designed multicenter study is needed to provide appropriate indications and guidelines for therapy in these patients.

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Discussion

Zieliński: I am a member of the European group investigating effects of nocturnal desaturation on pulmonary hemodynamics and survival. We investigated[1] the relationship between pulmonary function data, sleep-related desaturation, and daytime pulmonary hemodynamics in a group of 94 COPD patients not qualifying for conventional oxygen therapy (daytime arterial oxygen tension in the range 56–69 mm Hg). At entry, patients were divided into desaturators and nondesaturators. Desaturators were those who spent $\geq$ 30% of the overnight pulse oximetry recording with $S_{aO_2} < 90\%$. An obstructive sleep apnea syndrome was excluded by polysomnography.

Sixty-six patients were desaturators (Group 1) and 23 were nondesaturators (Group 2). There was no significant difference between the groups with regard to pulmonary volume or P_{aO_2} (63 $\pm$ 4 vs 63 $\pm$ 3 mm Hg), but P_{aCO_2} was higher in Group 1 (45 $\pm$ 5 vs 40 $\pm$ 4 mm Hg; $p < 0.0001$). Mean pulmonary artery pressure was very similar in the two groups (19 $\pm$ 5 vs 19 $\pm$ 4 mm Hg). No individual variable or combination of variables could predict the presence of pulmonary hypertension in an individual patient.

Next, we randomly divided patients with nocturnal desaturation into nocturnal oxygen treatment (NOT) and control groups. Both groups were observed for (mean) 39 months. Five of the 31 patients receiving NOT and 6 of the 25 control patients died. Difference in mortality was not significant. After two years of observation, the pulmonary hemodynamics study was repeated. The change in mean pulmonary artery pressure in NOT patients was +1.1 $\pm$ 4.2 mm Hg vs +1.8 $\pm$ 4.1 in controls (not significant). Those preliminary results suggest that NOT does not prolong survival in patients with moderate hypoxemia and does not decrease pulmonary arterial pressure.[2]

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O'Donohue: So the bottom line is it's very easy to put nocturnal desaturation patients on oxygen therapy in the United States. All you have to do is demonstrate that at any time during sleep the saturation is 88% or less. But there is very little definitive evidence of benefit from any studies done either here or in Europe.

Pierson: Walter, I have a question about logistics and money. You gave us indications for reasonably selecting patients with NOD who would perhaps be treated with oxygen therapy. If your figure of 14 million COPD patients in the United States is correct, and even allowing that all one million people on home oxygen that Tom (Petty) tells us about are desaturated during the day, that still leaves 13 million people who might potentially be candidates for NOD and who would thus need a sleep study.

O'Donohue: Today, obviously, you don't have to do a sleep study except for a saturation measurement in order to qualify for nocturnal oxygen in the United States. But based on the data that we've presented, in order to document changes during REM sleep, there's no question that you would have to have a formal sleep study.

MacIntyre: Do you test all COPD patients for nocturnal S_{pO_2}, and if not, who do you test? If you find someone who desaturates, do you evaluate the right heart before prescribing nocturnal oxygen?

O'Donohue: First of all, if a patient is not hypoxemic while awake, I would not do anything unless I had evidence of tissue hypoxia that I couldn't explain, for instance (and we see this not too infrequently) if a COPD patient is found to have evidence of right-sided heart failure with edema or an elevated hematocrit and his or her awake P_{O_2} is 60 mm Hg or above. In these patients, I would look for nocturnal desaturation. But most of these patients will not have NOD as we've defined it; rather they will have other, more severe sleep disturbances. Often they will have obstructive sleep apnea.

MacIntyre: So you start with the symptoms and then do the sleep study as opposed to the other way around.

O'Donohue: Yes, I would not monitor every COPD patient whose P_{aO_2} is above 60 mm Hg to see whether they desaturated at night, because I wouldn't know what to do with that finding alone, anyway. I would not start them on oxygen therapy unless they had signs of tissue hypoxia, and then the concern is that it may not be NOD alone that is causing nocturnal desaturation. It's possibly a more severe sleep disturbance.

Zieliński: I completely agree that we should look at symptoms first and then do the sleep study. In the study I was talking about, the only functional variable that could predict, in a way, the nocturnal desaturation was hypercapnia. So if the patient with P_{aO_2} above 60 mm Hg has hypercapnia, perhaps in this patient we should perform at least pulse oximetry during the night.

O'Donohue: I would agree, but I would also add that most of my COPD patients who are hypercapnic are also hypoxemic during the day, so the two tend to go together. It's very rare that I see a COPD patient who has significant hypercapnia but is not also hy-

poxemic breathing room air when awake.

Ronda Bradley:* When you test symptomatic patients, those with an increased hematocrit or the evidence of right-heart failure, do you typically go directly to a full polysomnography or would you do an overnight oximetry sleep study first?

O'Donohue: My co-author would probably disagree with me because of her interest in sleep studies. We frequently do a nocturnal trend-ox study, and if we find that the patient desaturates, supplemental oxygen is administered to see whether the desaturation is corrected by oxygen alone. Clearly, if it's not corrected with oxygen alone, the patient needs a formal sleep study. My co-author would say that all of these patients should probably have a sleep study.

Pierson: I want to ask Walter and the group a question that I don't think is going to come up in other contexts. The question is about polycythemia. I talked about chronic mountain sickness, the hallmark of which is profound polycythemia and, as I said, these people—for whom chronic hypoxia is the mechanism of their disease after all—have hematocrits always up in the 60s and often way up into the 70s. It seems to me that polycythemia (or erythrocytosis) is a very prominent part of cor pulmonale in the past, but it doesn't seem like we see it very much anymore. Why do you think that is?

O'Donohue: I think, first of all, that many of these people have multisystem disease that keeps their red blood cell mass reduced, or they may have a relative anemia of chronic disease. But I also think that more and more of the hypoxemic patients are receiving oxygen therapy earlier and never develop polycythemia (or erythrocythemia). Also, there is one other interesting phenomenon observed in transtracheal oxygen studies. In the Denver COPD patients (more so than in our patients, who live close to sea level), there were a number of patients who had been receiving nasal oxygen for more than 2 years who still had elevated hematocrits.[1] When they received transtracheal oxygen, the hematocrits fell. So even with "usual" oxygen therapy there are some patients who have enough hypoxemia (that is 5 or 6 hours daily that they're not using the oxygen) to maintain the elevated erythrocytosis in Denver. With continuous oxygen therapy by transtracheal catheter this can be corrected. I would agree with Dave (Pierson); particularly at or near sea level I see very few people who have erythrocytosis.

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Pierson: Of all the COPD patients I have, all of whom I try to get to use their oxygen and so forth, I don't think any one of them has a hematocrit over 55 in my practice.

Wedzicha: That is very interesting. This is exactly the same observation we found in the United Kingdom. I think there are a few factors. I don't know whether you've got the same observations, but we're seeing less blue and bloated patients; they're more in the pink and puffing range. These are the people who didn't have polycythemia before. The other factor, I think, is smoking. I think there's much less cigarette smoking going on in these people. They're more encouraged to stop, for safety reasons. Smoking was a very important determinant of erythropoietin and, thus, polycythemia.

Pierson: I have a theory about this. Maybe we don't see much secondary polycythemia in our hypoxic COPD patients because we prevent it with theophylline. A study in the *New England Journal of Medicine* several years ago showed that theophylline suppressed erythropoietin production in post-renal transplant with erythrocytosis as well as in normals,[1] and a later study showed that erythrocytosis could be reversed with theophylline in many post-renal transplant patients.[2] It was also shown recently that COPD patients on theophylline had lower hematocrits than those who were not taking the drug.[3] I wonder if, because of the almost universal use of theophylline in COPD, at least until recently in the United States, we didn't prevent polycythemia by that mechanism.

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Zieliński: There may be another factor involved. There were studies done on erythropoietin release during sleep in hypoxemic patients. The hypoxemia should last more than two hours with $P_{aO_2} < 60$ mm Hg in one run to trigger erythropoietin release.[1]

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O'Donohue: I think that helps make the point that in COPD patients with NOD who are not hypoxic while awake, you're not likely to see erythrocytosis, because they don't have severe and prolonged hypoxemia during sleep.

*Rhonda Bradley, Mallinckrodt Inc, St. Charles, Missouri.

Oxygen Therapy and Exercise Response in Lung Disease

Neil R MacIntyre MD FAARC

Introduction: The Normal Exercise Response
Can Supplemental Oxygen Improve Exercise Performance in the Normal Human Being?
Mechanisms of Exercise Impairment in Lung Disease
Lung Disease Results in Arterial Hypoxemia
Ventilatory Muscle Overload Increases Arterial Carbon Dioxide Tension, Resulting in Exercise-Limiting Acidosis
Impaired Cardiovascular Function Impairs Oxygen Delivery
Other Factors
Can Supplemental Oxygen Improve Exercise Performance in Lung Disease?
Can Long-Term Supplemental Oxygen Affect Long-Term Exercise Performance in Chronic Lung Disease Patients?
Summary

[Respir Care 2000;45(2):194–200] *Key words: oxygen therapy, exercise performance, exercise impairment, lung disease, hypoxemia, cardiovascular, metabolism.*

Introduction: The Normal Exercise Response

The normal cardiorespiratory response to exercise is summarized in Figure 1. As can be seen, when work load is incrementally increased, the cardiorespiratory system is called upon to deliver increasingly higher amounts of oxygen and to clear increasingly higher amounts of carbon dioxide.[1,2] In the early stages of exercise, aerobic metabolism predominates and the relationship of carbon dioxide production ($\dot{V}_{CO_2}$) to oxygen consumption ($\dot{V}_{O_2}$) (the respiratory quotient [RQ] value) is roughly 0.8. At higher levels of exercise, oxygen delivery is no longer adequate to aerobically meet metabolic demands and anaerobic metabolism begins to develop within exercising muscle cells. The resulting lactic acid production increases $\dot{V}_{CO_2}$ relative to $\dot{V}_{O_2}$ such that RQ rises above 1.0.

Since oxygen delivery is the product of cardiac output and arterial blood oxygen content, it is readily apparent that the heart and the lungs must work in concert to meet these metabolic demands. The cardiovascular system responds with increasing cardiac output, initially by an increase in stroke volume and then by an increase in heart rate.[1,2] Indeed, it is the cardiovascular component of oxygen delivery that limits exercise in normal human beings. The normal cardiovascular response is a 5-fold increase in cardiac output, which is accomplished by a near-doubling of the stroke volume and an increase of heart rate to approximately 220 minus the subject's age.

The ventilatory system has more reserve than the cardiovascular system and is thus not the rate-limiting step to exercise and oxygen delivery in a normal situation. Specifically, alveolar ventilation can increase almost 20-fold, and this is more than enough to meet oxygen and carbon dioxide transport demands during the most strenuous exercise.[1,2] Like the heart, the ventilatory response is initially an increase in tidal volume, followed by an increase in ventilatory rate. The normal ventilatory limit at maximum exercise is on the order of 60–70% of the 15-second maximum voluntary ventilation.[1,2]

The ventilatory response is determined primarily by pH. This produces a two-phased ventilatory response to exercise: an initial rise in exercise ventilation determined by the need to clear carbon dioxide from aerobic metabolism, and, later, a second, more rapid rise in exercise ventilation

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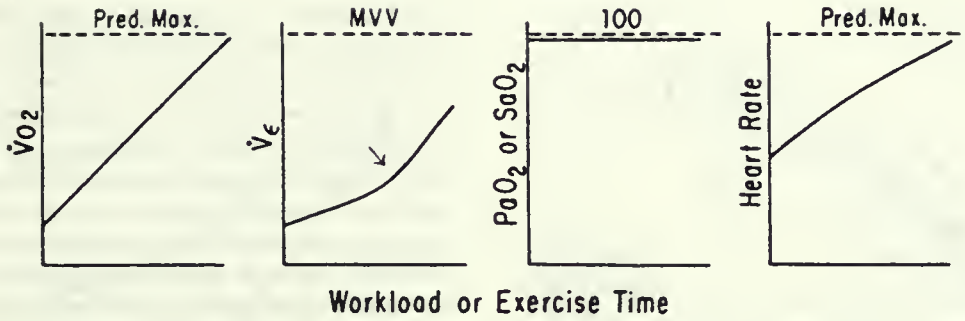

Fig. 1. Schematic representation of the normal response to exercise. The horizontal axis for all panels is work load. The first panel shows oxygen consumption ($\dot{V}_{O_2}$), which rises linearly to a predicted maximum. The second panel shows exercise ventilation ($\dot{V}_E$), which rises to match carbon dioxide production ($\dot{V}_{CO_2}$) and maintain pH homeostasis. When anaerobic metabolism increases the rate of rise of $\dot{V}_{CO_2}$, the $\dot{V}_E$ rate of rise also increases. This change in the $\dot{V}_E$ slope is termed the "anaerobic threshold" (arrow). Note that $\dot{V}_E$ does not reach the maximum voluntary ventilation (MVV) under normal circumstances. The third panel shows exercise arterial partial pressure of oxygen (P_{aO_2}). Because $\dot{V}/\dot{Q}$ relationships remain near unity throughout normal exercise, P_{aO_2} remains constant. The fourth panel is heart rate. As exercise increases, the cardiovascular system responds with increases in stroke volume and heart rate. Indeed, it is the cardiovascular system reaching its maximum (reflected by reaching maximum predicted heart rate) that limits exercise in normal subjects.

to clear the additional carbon dioxide from anaerobic metabolism. This changeover from the first phase to the second phase can be appreciated from inspection of the ventilation response to exercise, and is termed the "anaerobic threshold" (see arrow in Fig. 1).[1,2]

Gas exchange is well maintained during exercise in normal subjects. This is because ventilation-perfusion ($\dot{V}/\dot{Q}$) relationships remain near unity even under heavy exercise conditions.[3] The increased perfusion, however, increases the speed with which the red cells pass through the capillaries.[4] Hemoglobin exposure to alveolar gas (transit time) thus drops from approximately one second to less than half a second at high levels of exercise. This reduced transit time, however, is not enough to impact oxygen and carbon dioxide transfer in the setting of a normal alveolar-capillary interface. The net effect of normal $\dot{V}/\dot{Q}$ matching and adequate alveolar-capillary transport is that the arterial partial pressure of oxygen thus remains normal throughout exercise in a normal situation.

Can Supplemental Oxygen Improve Exercise Performance in the Normal Human Being?

Since the early 1900s there have been a number of anecdotes reporting that supplemental oxygen improves exercise performance.[5,6] Indeed, a famous runner, Roger Bannister, in 1953 wrote an extensive report saying that supplemental oxygen improved endurance in athletes.[7] Many of the early experiments, however, were not blinded or randomized. In 1952 Miller made the observation that if the subject was told the breathing gas was supplemental oxygen (even though it was room air), the subject's exercise performance improved.[8]

More carefully done studies in the latter half of the 20th century, however, do suggest that hyperoxia can improve maximal oxygen consumption to a small degree (generally $< 10\%$).[9] Attempts have been made to separate the effects of cardiac output and oxygen content in these observations. In general, the weight of evidence is that hyperoxia has little if any effect on cardiac output at maximal exercise. In contrast, however, hyperoxia can be shown to improve oxygen content, primarily through increases in dissolved oxygen in plasma (again generally $< 10\%$).[9] Interestingly, despite small increases in oxygen delivery, hyperoxia seems to cause a *decrease* in regional blood flow to exercising muscles.[10] The net effect, then, is that regional oxygen delivery to exercising muscles during supplemental oxygen is probably negligible.

Another explanation for an increase in exercise capacity during hyperoxia involves cellular metabolism.[9] A number of studies have shown that under similar exercise loads, hyperoxia reduces lactate production.[9,11,12] Hyperoxia also reduces RQ, resulting in less carbon dioxide production.[9,11] Possible mechanisms for these changes include hyperoxia-induced fatty acid metabolism, hyperoxia-induced prolongation of aerobic metabolism, and hyperoxia-induced respiratory acidosis (see below) reducing lactate production.

A consistent observation in many studies is that exercise under hyperoxic condition reduces the minute ventilation at a given level of exercise (Fig. 2).[9] This results in a mild respiratory acidosis at any given level of exercise, compared to that which would be seen on room air. The reason for this suppression of the ventilatory response is not clear but probably relates to a depression of the partial pressure of oxygen (P_{O_2}) related respiratory drive in the carotid bodies.[13] Indeed, this is comparable to the prolongation of breath-holding that can be achieved with supplemental oxygen breathing. The resulting mild respiratory acidosis (permissive hypercapnia) may have several effects that enhance exercise performance. The reduction in lactate

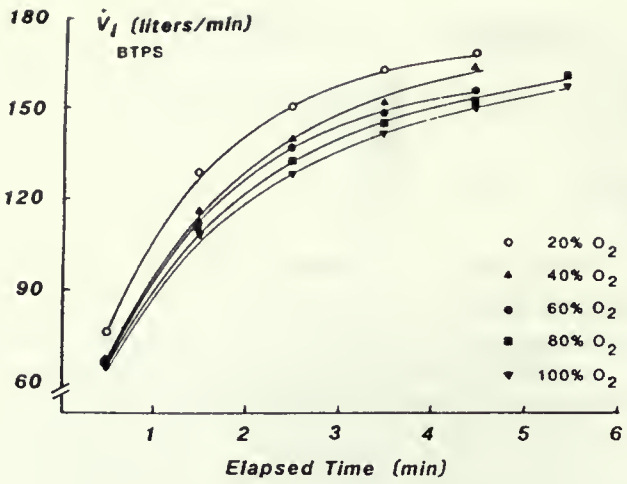

Fig. 2. Mean minute ventilation ($\dot{V}_I$ = minute ventilation) as a function of exercise duration using 5 different inspired oxygen concentrations. Note the progressive drop in exercise ventilation as higher concentrations of oxygen are inspired. (From Reference 9, with permission.)

noted above may in fact be due to a mild buildup of intracellular acid.[12] An additional effect of this ventilation reduction is that ventilatory muscle work and resulting metabolic demands are mildly reduced.[9,14]

The overall conclusion, then, is that hyperoxic gas mixtures seem to have a small but real effect on exercise performance in normal human beings. Interestingly, however, much of this effect is related to the development of a permissive hypercapnia through suppression of oxygen-related ventilatory drive and has little to do with increases in actual oxygen delivery to the tissues.

Mechanisms of Exercise Impairment in Lung Disease

Lung disease can affect the exercise response through a number of different mechanisms.[15–21] These are summarized below.

Lung Disease Results in Arterial Hypoxemia

Hypoxemia from lung disease has a number of mechanisms.[17,18,21] First, impaired ventilation reduces the alveolar P_{O_2}. This can be made worse with exercise when ventilatory muscles become overloaded and cannot provide appropriate ventilation. Most lung diseases have abnormal $\dot{V}/\dot{Q}$ relationships within the lung as a major cause of hypoxemia.[22] In many of these conditions, these $\dot{V}/\dot{Q}$ mismatches can worsen with exercise. Interestingly, however, this may not be the case in chronic obstructive pulmonary disease (COPD).[23] Lung diseases with shunts have hypoxemia at rest, but the response of a shunt to exercise depends upon the blood flow through the shunt with exercise. If exercise increases shunt flow, profound exercise hypoxemia results. In contrast, capillary recruitment away from the shunt may actually improve oxygenation with exercise. As with $\dot{V}/\dot{Q}$ mismatch, shunting in COPD is usually not an important mechanism of hypoxemia. Finally, the transit time of the red cell through the alveolar capillary is shortened during exercise, as noted above. While this is not an important issue in the normal lung, in an abnormal lung with diffusion abnormalities this reduced transit time may result in worsening hypoxemia.[3]

Ventilatory Muscle Overload Increases Arterial Carbon Dioxide Tension, Resulting in Exercise-Limiting Acidosis

Most lung diseases impose excessive loads on ventilatory muscles.[18,24] These loads can be in the form of worsening compliance from parenchymal disease or worsening airway resistance in the obstructive diseases. Under these conditions, ventilatory muscles can become overloaded as they try to meet the ventilatory response to exercise,[1,2,18,25] and this overload can precipitate muscle fatigue and failure, with a resulting respiratory acidosis. Ventilatory muscle overload develops quicker in the presence of wasted ventilation, especially when wasted ventilation increases with exercise (typical of pulmonary vascular disease). Finally, inadequate oxygen delivery coupled with intrinsic muscle dysfunction from disease further reduces the ability of the ventilatory muscles to properly clear carbon dioxide.[18,25]

An additional problem in the obstructive disease patient is the development of an inefficient ventilatory pattern with exercise.[25] This is characterized by tachypnea and a shortened expiratory phase, which leads to air trapping. This air trapping in turn puts the inspiratory muscles in a shorter configuration that further reduces the capacity to perform work.

Impaired Cardiovascular Function Impairs Oxygen Delivery

Both intrinsic lung disease and chronic hypoxemia result in pulmonary vascular abnormalities.[26–29] These in turn produce pulmonary hypertension that overloads the right ventricle. This impaired myocardium has less capability of meeting oxygen delivery demands, especially under exercise conditions. Compounding this can be dysrhythmias, which can develop in the face of hypoxemia and acidosis. Finally, deconditioning of the entire cardiovascular system can occur in patients who have chronic lung disease (CLD) and decreased activity. Simply put, patients "decondition themselves down to their level of dyspnea."

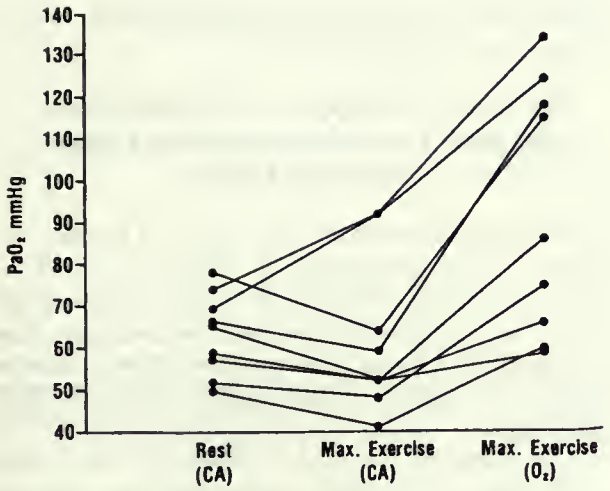

Fig. 3. Response of exercise hypoxemia to oxygen supplementation in 9 COPD patients. CA = compressed air. P_{aO_2} = arterial oxygen tension. (From Reference 32, with permission.)

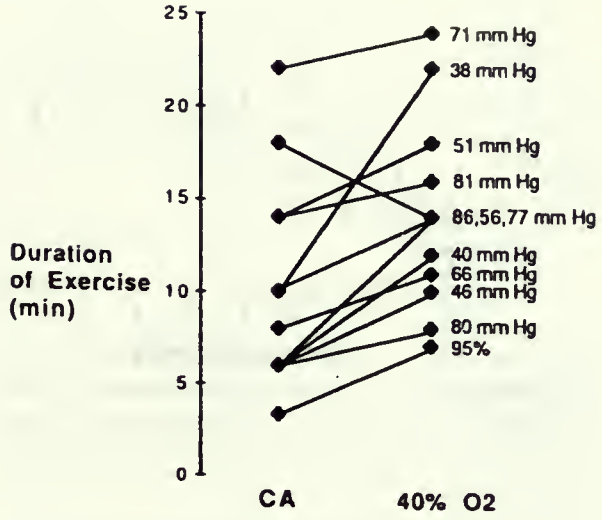

Fig. 4. Duration of exercise while breathing compressed air (CA) versus 40% oxygen in 11 COPD patients. The arterial oxygen tension or arterial oxygen saturation at maximum exercise while breathing CA is listed for all patients. Note that exercise improvement occurred in 10 patients while breathing oxygen, regardless of whether or not they had exercise hypoxemia during CA breathing. (From Reference 33, with permission.)

Other Factors

Patients with CLD often have a number of nutritional disturbances that can further impair exercise performance.[18] These patients can also have coexisting diseases, such as heart disease and peripheral vascular disease, which can impact exercise performance.[18] Psychological factors can also impair the exercise response, particularly in CLD patients with significant anxiety or depression. Finally, the perception of dyspnea resulting from abnormal thoracic stretch receptors, irritant receptors, and/or blood gas receptors may also limit a patient's ability to exercise.[18,30]

Can Supplemental Oxygen Improve Exercise Performance in Lung Disease?

In considering the causes of exercise impairment in CLD noted above, oxygen supplementation can conceivably improve performance in a number of ways.[18,23,25,29,30–35] First, if exercise hypoxemia occurs, supplemental oxygen can reverse this. Second, ventilatory muscle loads in ventilatory muscle function might be improved through several different mechanisms discussed below. Third, pulmonary vasoconstriction and dysrhythmias secondary to hypoxemia plus right heart dysfunction may be reduced to provide better cardiovascular function. Fourth, subjective dyspnea caused by hypoxemia-related receptors might be blunted. Each of these will be discussed separately.

Supplemental oxygen can clearly *reverse hypoxemia* that occurs with exercise in CLD (Fig. 3).[32] To the extent that hypoxemia impairs oxygen delivery during exercise in these patients, supplemental oxygen should be expected to improve performance. Indeed, numerous studies in patients with exercise hypoxemia have found improved performance when the exercise hypoxemia was prevented.[31–35]

A number of studies have quite clearly shown, however, that CLD patients who do not have exercise desaturation may also benefit from oxygen supplementation during exercise testing (Fig. 4).[33] The effects of supplemental oxygen on *exercise ventilation* may provide some of the explanation for this. First, Libby et al[36] have shown that in some patients with obstructive lung disease, supplemental oxygen reverses hypoxia-induced bronchoconstriction. This in turn can reduce some of the ventilatory muscle loads imposed during exercise. Perhaps more importantly, supplemental oxygen reduces the ventilation at a given level of exercise (Fig. 5).[32] Note that this occurs even if severe hypoxemia is not present, and resembles what is observed in normal subjects (see Fig. 2). Similar to normal subjects, this reduced ventilation and permissive hypercapnia may have beneficial effects on cellular metabolism as well as lowering ventilatory muscle work requirements.[11,12] Perhaps more important in the obstructive disease population, however, is the fact that the reduced ventilation is also accompanied by a prolongation of the expiratory phase.[23,25] This reduces the development of air trapping, thereby preventing extra loading on the ventilatory muscles. Ventilatory muscle function may also be improved and fatigue forestalled if oxygen delivery to them can be improved with supplemental oxygen.

If right heart dysfunction is present in patients with CLD, exercise may be impaired. Adding supplemental oxygen has been shown to *benefit right heart function* in

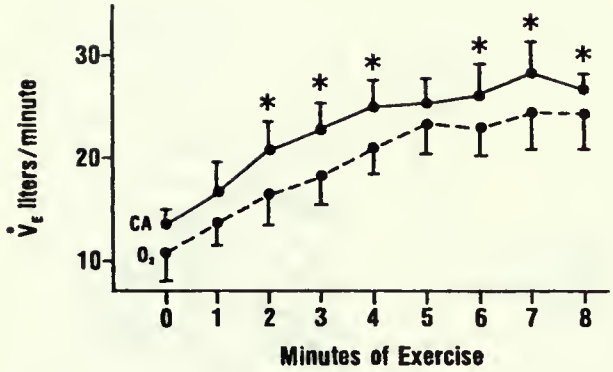

Fig. 5. Ventilation during increasing levels of exercise in COPD patients breathing compressed air (CA) or supplemental oxygen (O_2). Exercising with supplemental oxygen significantly reduced the ventilation. $\dot{V}_E$ = expired volume per minute. (From Reference 32, with permission.)

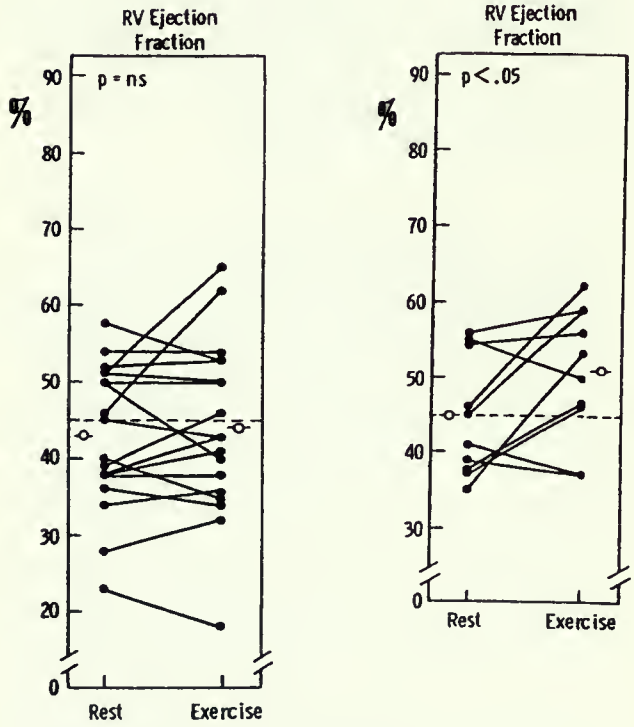

Fig. 6. Right ventricular (RV) ejection fraction during rest and exercise while breathing room air (left panel) and oxygen (right panel) in patients with underlying right ventricular dysfunction. In these patients the supplemental oxygen significantly improved the exercise response of the right ventricle. (From Reference 29, with permission).

those patients in whom right heart dysfunction is present (Fig. 6).[29]

Finally, the *reduction in dyspnea* resulting from supplemental oxygen may improve exercise duration,[31] (Fig. 7) although this is a controversial point.[37] As noted above, the mechanisms underlying this in the nonhypoxemic patient are unclear but may be related to attenuation of P_{O_2}-related carotid body activity.[38]

Can Long-Term Supplemental Oxygen Affect Long-Term Exercise Performance in Chronic Lung Disease Patients?

Given that supplemental oxygen in CLD patients can improve exercise performance as measured by exercise tests, two important questions follow. First, does long-term use of oxygen benefit patients who have only exercise hypoxemia? Second, can long-term use of oxygen as part of a rehabilitation program benefit long-term exercise performance?

The first question was recently addressed by McDonald et al,[39] who studied COPD patients who did not have resting hypoxemia but did have exercise hypoxemia. Six weeks of oxygen use in these patients offered small (but significant) benefits in terms of 6-minute walk test and quality-of-life score, but not in dyspnea score or exercise testing. Exercise training was not a part of this study.

The second question was recently addressed in an interesting study by Kramer et al.[40] This group studied COPD patients normally living in Jerusalem during rehabilitation at the Dead Sea. This environment change caused an increase of approximately 104 mm Hg barometric pressure and was equivalent to an additional 22 mm Hg inspired P_{O_2}. Interestingly, this rehabilitation program seemed to offer benefit that persisted even upon return to sea level (Fig. 8). Others have also suggested that supplemental oxygen can facilitate exercise training in hypoxemic COPD patients.[41,42]

In our own rehabilitation program we have the opportunity to categorize obstructive lung disease patients during exercise as being impaired by one or more of three factors: gas exchange impaired (arterial oxygen saturation measured via pulse oximetry S_{pO_2} during exercise $< 88\%$ or $P_{O_2} < 60$ mm Hg), ventilation impaired (exercise ventilation $> 80\%$ of the measured maximum voluntary ventilation), or cardiovascular impaired (maximum exercise heart rate $> 80\%$ of predicted). In obstructive lung disease patients who are only cardiovascular-limited (ie, not ventilation-limited or gas exchange-limited), a 20-session exercise program clearly improved the $\dot{V}_{O_2}$ and walking performance (Table 1, cardiovascular group). In contrast, the response to this program in patients with gas exchange limitations is much less impressive (see Table 1, gas exchange groups). As can be seen in Table 1, there are consistent trends toward improvement in these patients when they undergo exercise training with supplemental oxygen, but these differences are small and in many cases not significantly different than baseline. Thus, the role of oxygen and conditioning in COPD patients remains unclear and is a topic for future research.

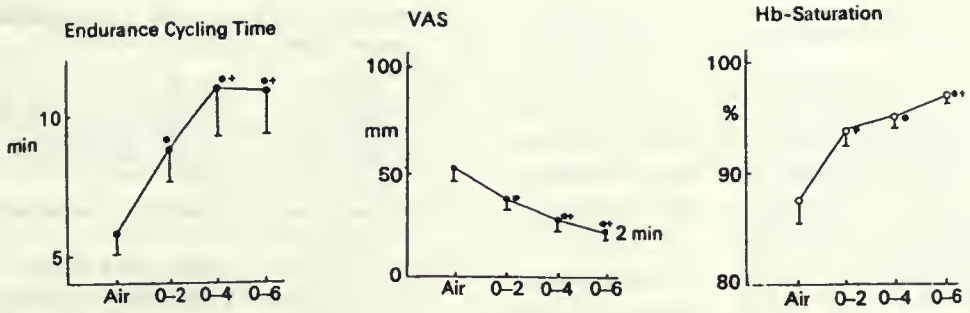

Fig. 7. Exercise duration (left panel), dyspnea perception at 2 minutes of exercise (middle panel) (VAS = visual analogue scale), and hemoglobin saturation with oxygen (right panel) in hypoxemic lung disease patients exercising while breathing room air (Air) and 3 different supplemental oxygen flows (2, 4, and 6 L/min). Note that exercise hypoxemia was eliminated by oxygen at 2 L/min and that exercise tolerance and dyspnea both improved with this. Note also that further increases in supplemental oxygen further increased exercise tolerance and reduced dyspnea. (From Reference 31, with permission.)

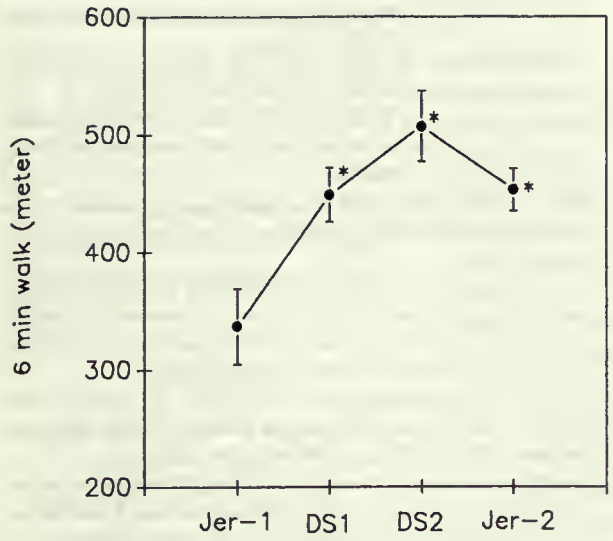

Fig. 8. Improvement in exercise performance after training hypoxemic COPD patients in a relatively hyperbaric environment (Dead Sea). Exercise performance is measured by the 6-minute walk test (vertical axis). Jer-1 represents baseline measurements at 800 m above sea level (Jerusalem). DS1 and DS2 represent measurements at the beginning and end of training at the Dead Sea (400 m below sea level), respectively. Note the conditioning effect after training and the persistent improvement even upon return to Jerusalem (Jer-2). (From Reference 40, with permission.)

Table 1. Mean Changes in Exercise Performance in COPD Patients after 20 Rehabilitation Sessions

Exercise Limiting Factor(s)	*n*	Pre $\dot{V}O_2MAX$ (mL/min)	Post $\dot{V}O_2MAX$ (mL/min)	Pre Laps Walked	Post Laps Walked
CV	183	917	1,062*	9.0	11.0*
O_2 alone	17	564	646	6.8	8.9
O_2 + V_e	23	592	639	5.0	6.9*
O_2 + CV	25	782	830	7.4	9.1*
O_2 + V_e + CV	22	736	736	6.3	8.2

COPD = chronic obstructive pulmonary disease; $\dot{V}O_2$ = oxygen consumption; CV = cardiovascular limit; O_2 = gas exchange limit; V_e = ventilation limit.
*$p < 0.5$ post compared to pre values.

Summary

Lung disease affects exercise performance through a number of mechanisms, including hypoxemia, abnormal ventilatory mechanics, abnormal ventilatory muscles, abnormal ventilatory patterns, abnormal right heart function and subjective dyspnea. Supplemental oxygen improves hypoxemia and thus improves exercise impairment resulting from hypoxemia-related reductions in oxygen delivery. Supplemental oxygen also reduces exercise ventilation. This, in turn, reduces ventilatory muscle work, and the concomitant permissive hypercapnia may have beneficial effects at the cellular level. Additionally, in obstructive disease patients, an improved ventilatory pattern may reduce air trapping. Supplemental oxygen may also improve right ventricular dysfunction in patients with underlying right ventricular dysfunction. Finally, supplemental oxygen may reduce dyspnea caused by oxygen-related carotid body activity.

Important questions remain. First, is long-term oxygen use of benefit in patients with only exercise hypoxemia? Second, is exercise conditioning possible in patients with exercise hypoxemia? Third, does supplemental oxygen enhance exercise conditioning efforts in those patients with CLD but without exercise hypoxemia? If the answer to this last question is yes, what selection criteria should be used to identify those who would benefit? The answers to all of these questions will have enormous impact on our approach to the optimal management of CLD patients.

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Discussion

Stoller: Neil, one of the things you didn't talk about is the ascertainment of desaturation during exercise, vis-à-vis pulse oximetry versus arterial blood gases. Dean (Hess) showed us Brian Carlin's data about the assessment of criteria for supplemental oxygen at rest, arguing that pulse oximetry, with its 3–5% variation from arterial oxygen saturation measured via blood gas analysis, can be misleading.[1] My sense is that this is even more confounding during exercise. I'd be interested in your thoughts with regard to the practical question of a normoxemic patient for whom you're trying to make a decision about supplemental oxygen during exercise. How should one do that? How should one decide whether oxygen desaturation is occurring, and then how should one decide about the dose of supplemental oxygen to provide in that setting?

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MacIntyre: Well, that's a very important point, and I didn't bring my data here to show the correlation between exercise S_{pO_2} and P_{aO_2}. We have that data. It's a good correlation, but as you suggest, and as Dean clearly showed, there is scatter there. Thus, in my laboratory, in my rehabilitation program, we will not diagnose exercise hypoxemia unless we demonstrate it with an arterial blood gas. We measure arterial blood gases and S_{pO_2} simultaneously while the patient exercises on a bicycle, and from this we get the correlation between those two measurement techniques in that particular patient, and use that to guide oxygen therapy during the rehabilitation process, using just pulse oximetry. So it's a key point and I think there's a potential for over- and under-diagnosis if you use pulse oximetry. In the National Emphysema Treatment Trial, which uses exercise as one of the end points, there are two things that the trial does that I found to be quite interesting. First, they do an oxygen titration measurement using S_{pO_2} with exercise. The patient serves as his/her own control and it's an end point in the study. The second exercise end point that we have in the National Emphysema Treatment Trial is the full cardiopulmonary exercise tests with all patients receiving 30% oxygen. Exercise hypoxemia is thus not an issue with this test.

Wedzicha: Neil, I think that was the most clear account I have ever heard of exercise hypoxemia. Short-burst oxygen treatment is a problem in the United Kingdom. All that means is using oxygen cylinders for a few puffs for a few minutes here and there. There is a lot of short-burst oxygen used in the United Kingdom. Although you showed us elegantly that patients who do not desaturate during exercise may get a benefit, I think there's very little evidence that the occasional use of oxygen before, after, or at recovery from exercise actually has much effect.

MacIntyre: You are speaking of "prn oxygen." Walter and I have been sort of laughing about that all along during this conference. It's never really made a lot of sense to me. It seems to me it ought to be used for physiologic reasons. As you point out, however, there are plenty of patients who just kind of puff on it when they feel anxious or dyspneic. Another group is the athletes who, on the sidelines, believe that a whiff or two of oxygen from time to time is a good idea. I don't know how well it's caught on in the United States, but in Toronto, Canada, they have oxygen bars where you can go in and hook yourself up to a couple liters a minute and socialize with your friends.

Pierson: They're big in Japan, too.

MacIntyre: One study I did not show because of time limitations looked at oxygen during exercise recovery in COPD patients. Exercise recovery in normals is probably not affected by supplemental oxygen, but there is at least one study I'm aware of where the sense of recovery and the return to a baseline of subjective measurements was considerably shortened with the use of oxygen in COPD.[1] So perhaps in the recovery phase of exercise there may be some benefit. But I would agree with you that prn oxygen doesn't make a whole lot of sense.

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Wedzicha: The other point is the use of oxygen during rehabilitation. Rooyackers et al investigated this.[1] The problem with their work was that the patients did not desaturate on exercise. We have completed a study looking at the use of supplemental oxygen in desaturators during an 8-week rehabilitation program.[2] Patients used oxygen only during the program in hospital. Our results showed relatively little benefit. We did, however, find a decrease in dyspnea in the oxygen-treated rehabilitation exercise-trained group.

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MacIntyre: But they were not desaturators?

Wedzicha: Everybody was a desaturator in the study.

MacIntyre: The exercise-limiting factors in COPD patients, I think, are important in predicting the exercise response. One of the problems with all the exercise literature in COPD is that it tends to group all COPD patients into a single entity. The way we've approached it in our program is we look for the specific factors that limit people. Are they pure desaturators? Is ventilatory response a key factor? Is cardiovascular response out of whack? Or is it some combination of all three of those? I think in trying to sort out what exercise and/or oxygen is going to do, it's very important to categorize what the major limiting factors are in the exercise response. To be honest, I'd have to sit down for a while with you and your data to sort out which group was doing what to make any sense out of it. I think it is a very complex field. All COPD patients are not the same when it comes to exercise response.

Greg Spratt:* From the data that was presented earlier by Dr O'Donohue and from what you're presenting here on exercise, in patients who are hypoxemic at rest, oxygen needs are likely to increase during sleep. And, if I'm understanding your data correctly, increasing oxygen flow during exercise gives better results: endurance is longer. Yet, in practice, the prescription rarely reflects the need for increased oxygen usage during sleep and/or exercise. Do you feel there is a need to put more emphasis here? Is that important to address?

MacIntyre: I think you're exactly right. My whole outpatient practice really revolves around taking care of these folks in the rehab program. We have a lot of COPD patients, and people with other types of disease as well, but COPD is clearly our number one.It's very obvious that some of these folks come in and don't need oxygen at rest, but do desaturate with exercise, and we titrate them. Some come with oxygen at rest and get worse with exercise, and they need more oxygen with exercise. But there are some who actually improve with exercise and may not need as much. So I would agree that oxygen administration does require individual assessment—patients shouldn't all be treated the same way—and dose changes with exercise need to be considered. It goes back to the use of the devices Bob described. Just sort of grouping everyone at 2 L/min and sending them on their way is a gross oversimplification. I think there are dosing and device considerations in a number of circumstances. One of the pleas of this conference should be that reimbursement rates for proper assessment of oxygen, both in terms of dosing and devices, ought to be made available, so that the right people can be doing this sort of thing.

Greg Spratt: Would you suggest that a way to do that, typically, is pulse oximetry, titrate, and maintain saturation above 90% during those various times?

MacIntyre: We've already talked about the limitations of pulse oximetry, but having admitted that there are limitations, it's still not a bad approximation of what's going on. We could argue about whether the threshold should be 90% or 88% or 86%, but I don't think it's unreasonable to have a careful assessment of people under a variety of circumstances to set up their oxygen titration properly.

O'Donohue: To that point, I personally do not start any patient on long-term oxygen therapy without titrating oxygen flow for both rest and exercise. One of the problems with exercise is that standard exercise tests really do not predict whether patients desaturate with activities of daily living . Italian investigators have shown that a 6-minute walk test does not correlate with desaturation with activities of daily living.[1] So, basically, whenever a patient is started on long-term oxygen therapy, I have the patient walk at his or her own pace and determine (using pulse oximetry, and realizing its limitations) the oxygen flow needed to correct the hypoxemia. For most patients, walking at their own pace is typical of their usual activity of daily living. This is particularly important now with the greater use of oxygen conserving devices. The differences between the oxygen flow at rest and the flow with exercise may be magnified with those devices. Barry Make and associates have shown, particularly with transtracheal oxygen, reductions in inspiratory minute ventilation,[2] and Bartolome Celli et al have found a reduction in work of breathing.[3] It has been shown, even with nasal oxygen, that at higher flows there may be an "insufflation ventilation" effect. Would you speak to that, Neil?

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MacIntyre: I knew we'd get into mechanical ventilation here. You're speaking to the concept of tracheal gas insufflation, right? The principle is that during and at the end of the expiratory phase, the dead space in our airways has residual gas laden with carbon dioxide, and the idea of tracheal gas insufflation is that during the expiratory phase, a slow flow of fresh gas (oxy-

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gen) blows that carbon dioxide out of the dead space so that the next inspiration has less carbon dioxide in it. The dead space has been effectively reduced. In mechanical ventilation, there are a number of techniques to do this—putting catheters down at the trachea and blowing 8–10 L/min out during the expiratory phase. What Walter's speaking to is that the same phenomenon may occur from a nasal flow of gas, or, more effectively, probably, from a transtracheal flow of gas, where you put continuous flow in. Then, during expiration, it washes out the dead space. You're exactly right, Walter: a mechanism to reduce dead space and reduce ventilatory load would be to wash out dead space with tracheal gas insufflation.

O'Donohue: Reducing inspiratory minute ventilation?

MacIntyre: Sure, it makes ventilation more effective because you've reduced dead space.

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Ambulatory Oxygen Therapy, Exercise, and Survival with Advanced Chronic Obstructive Pulmonary Disease (The Nocturnal Oxygen Therapy Trial Revisited)

Thomas L Petty MD FAARC and Peter L Bliss BME

Introduction
Re-examination of the Nocturnal Oxygen Therapy Trial
Walking Information
Data Analysis
Results
Discussion
Summary

[Respir Care 2000;45(2):204–211] *Key words: nocturnal oxygen therapy, long-term oxygen therapy, chronic obstructive pulmonary disease, oxygen transport, ambulation, tissue oxygenation, exercise.*

Introduction

In the 1950s, Alvan Barach became a champion of ambulatory oxygen.[1] He developed a small cylinder for compressed gas for ambulatory oxygen, illustrated in Figure 1. Figure 2 is a cartoon designed by Barach. This illustration encourages patients to get going and to walk as they pursue the adventures of life. At about the same time, Cotes reported on his observations in ambulatory oxygen.[2] "The increased exercise ability when the patient is breathing oxygen is probably due to a reduction toward the normal levels of ventilatory requirements for exercise, as a result of the relief of anoxia." It was Cotes's conclusion that "Portable oxygen should be regarded as an integral part of domiciliary oxygen therapy."[3] At the time of that writing, he had been using oxygen successfully in 4 patients at home. Figure 3 shows how he transfilled a small high-pressure cylinder from a larger tank. Figure 4 shows two lightweight, high-pressure cylinders used by Cotes in his studies.

In the early Denver study, cited in the "Historical Highlights of Long-Term Oxygen Therapy," (Respir Care 2000; 45[1]:29–36) the most striking observation was a dramatic improvement in exercise tolerance during the oxygen administration month followed by the control month in two patients (Fig. 5).[4] It could be argued, however, that this exponential improvement in exercise tolerance during the month those patients received oxygen was simply a continuation of the results of exercise training during the control month while breathing air. However, the slope of the improvement is much steeper in the oxygen month.

Pierce and Miller showed reduced recovery time with oxygen-supported exercise, compared with oxygen breathing air.[5] Liker et al were the first to conduct a controlled double-blind trial comparing portable liquid oxygen with liquid air.[6] In their study all patients reported feeling better while carrying the portable device. Three of 9 patients showed a clear-cut increase in the distance walked while breathing oxygen, compared with air. This was a very difficult study to conduct, but it was important because it gave evidence that oxygen offered more than placebo value. Additional controlled exercise studies were conducted by Bradley et al[7] and by Leggett and Flenley,[8] among others. Bradley's group showed that walk endurance was significantly increased by oxygen, although the maximum work rate could not be equally improved.

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This paper uses data supplied by the National Heart, Lung, and Blood Institute, the National Institutes of Health, and the Department of Health and Human Services. The views expressed in this paper are those of the authors and do not necessarily reflect the views of the National Heart, Lung, and Blood Institute.

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Fig. 1. The late Alvan Barach modeling a small high-pressure oxygen cylinder capable of being transfilled from a large compressed gas L or K cylinder.

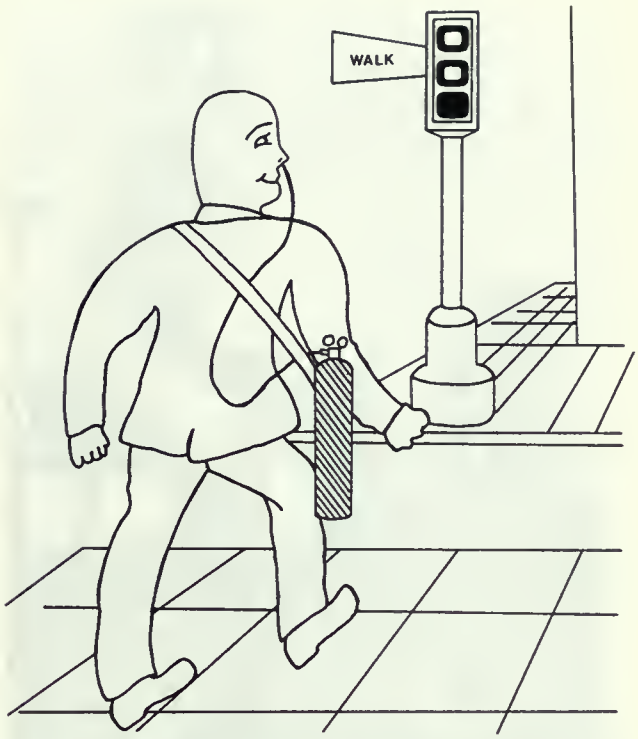

Fig. 2. Cartoon diagram by Dr Barach emphasizing the importance of walking with ambulatory oxygen.

The major purpose of this report on ambulatory oxygen is to offer a new analysis of the Nocturnal Oxygen Therapy Trial (NOTT) data in order to explore why continuous oxygen therapy (COT) was superior to nocturnal oxygen therapy (NOT). We aimed to determine if survival was related to the exercise capacities of the patients at the time of randomization to oxygen or if it was a function of the duration of oxygen administration.

Re-examination of the Nocturnal Oxygen Therapy Trial

In brief review, the NOTT study collected extensive data from a well-defined population of patients with advanced chronic obstructive pulmonary disease (COPD), who were randomly assigned to receive either NOT for approximately 12 hours per day from a stationary source or ambulatory oxygen (ie, COT that was intended to be used as close to 24 hours per day as possible). In the NOTT, extensive outcome data were obtained that can answer important questions about oxygen, exercise, survival, and hospitalization requirements in this well-defined population.

The details of the original NOTT study have been reported elsewhere.[9] Patients with chronic stable hypoxemia with partial pressure of oxygen (P_{O_2}) of $\leq$ 55 mm Hg who had no significant co-morbidities and were willing to participate in an exercise-oriented rehabilitation program utilizing oxygen were randomized to receive either NOT from a stationary oxygen system or COT from an ambulatory system.[9,10] The randomization process resulted in patients who were well-matched by age, gender, and indices of disease severity.[9] After the NOTT report, the magnetic data tapes were placed in the public domain in hopes that further analyses would be made by other investigators. This goal was not achieved at first because of the complexities of the data methods originally used, which were state-of-the-art computer technology at the time of the study.[11,12]

Recently, one of us (PLB), using newer software, was able to convert the original data into a personal computer format. The original NOTT data set was obtained from the National Heart, Lung, and Blood Institute in its original format and converted into an Access (Microsoft Corporation, Redmond, Washington) database. From this database, information was output to Excel (Microsoft Corporation, Redmond, Washington) for matching and analysis.

Fig. 3. JE Cotes transfilling oxygen system. (From Reference 2.)

Fig. 4. Two small high-pressure cylinders used by JE Cotes. (From Reference 2.)

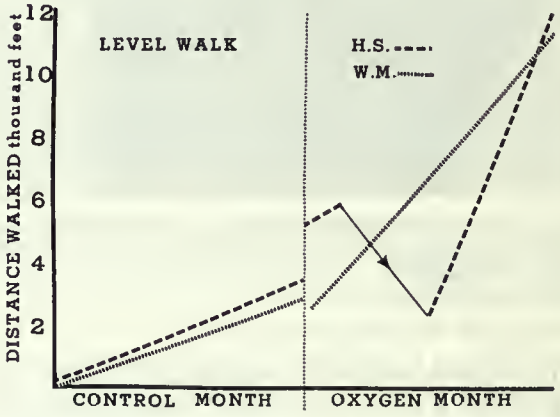

Fig. 5. Improvement in exercise tolerance in two patients trained at level walk. Note slow change during control month and marked rapid rise in tolerance during oxygen month. "H.S." and "W.M." are patients' initials. Temporary decrease in H.S.'s exercise tolerance during the oxygen month was due to an episode of acute bronchitis. (From Reference 4.)

Walking Information

In the NOTT, each candidate for the study was given a comprehensive pulmonary rehabilitation program, with exercise performed on a daily basis for 3 weeks prior to randomization to NOT or COT. Each patient was urged to walk as much as possible each day during the 3-week stabilization period, and each was given a pedometer to monitor the distance walked each day. This distance was recorded by a research nurse or technician. This 3-week

Table 1. Matching Data for Baseline Walking Comparison.*

Characteristic	Low Walk NOT	Low Walk COT	High Walk NOT	High Walk COT
n	22	18	22	18
Age, median, years	67.6	66.5	67.6	66.5
P_{O_2}, mm Hg	52.3	49.9	51.6	51.6
P_{CO_2}, mm Hg	45.7	42.6	42.4	43.2
pH	7.40	7.40	7.42	7.40
Heart rate, min^{-1}	92.6	95.2	85.9	95.8
Pulmonary artery mean pressure, mm Hg	31.6	29.1	26.9	30.1
Cardiac index, $L/min \cdot m^2$	2.76	2.88	2.62	3.04
Pulmonary vascular resistance, $dyn \cdot s \cdot cm^{-5}$	381	363	383	379
FEV_1, % of predicted pre-bronchodialator	25%	28%	25%	28%
FVC, % of predicted pre-bronchodialator	48%	51%	50%	49%

*Cardiac and blood gas data taken at rest with supplemental oxygen. All values are mean unless noted.

NOT = nocturnal oxygen therapy; COT = continuous oxygen therapy; FEV_1 = forced expiratory volume in the first second; FVC = forced vital capacity.

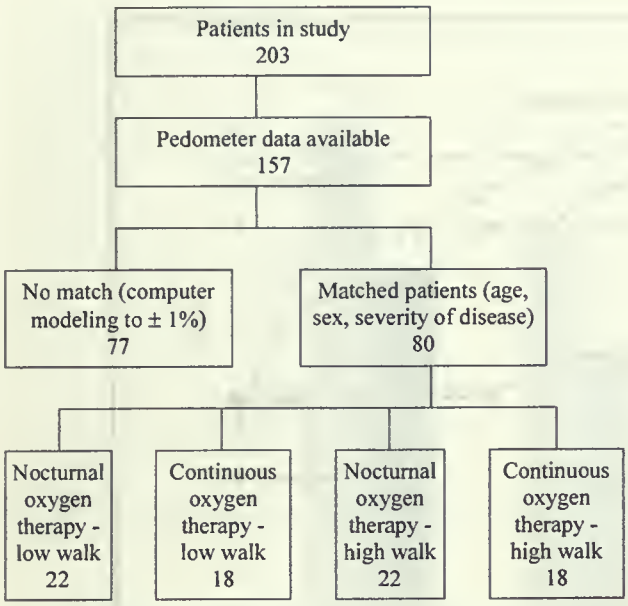

Fig. 6. Re-examination of the Nocturnal Oxygen Therapy Trial by pretreatment ambulation status. Origins of the 80 matched patients.

period was intended to bring each patient to a functional optimum before randomization to NOT or COT. The average number of feet that each patient walked per day was calculated during the third week of the stabilization period. We call this factor "walking level." Pedometer data for the 3-week stabilization and optimization period was available for 157 of the 203 total patients. We had maximum oxygen consumption measurements for 106 of the 203 total patients enrolled in the NOTT. In our matched group of 80 subjects we have maximum oxygen consumption measurements for 18 subjects with a low walking level (39 mL/min), and 24 subjects with a high walking level (59 mL/min) ($p = 0.007$). Thus, the walking level can be taken as a surrogate indicator of the maximum oxygen consumption measurement in this analysis.

We divided the patients into a low walking and a high walking group by separating at the median walking distance achieved at the end of 3 weeks. Figure 6 presents the origins of the 80 matched patients. The initial division of the patients by walking level at the median (3,590 ft/d) yielded groups that were not well matched by age. The low walking group was approximately 5 years older than the high walking group. To overcome this weakness, each low walking patient was manually matched with a high walking patient of similar age, treatment group (COT vs NOT), and percent of predicted forced expiratory volume in the first second. The resulting groups were 40 patients each in the low and high walking groups. These 80 patients were then further divided into COT and NOT groups. Table 1 shows the results of this matching, and this shows good matching by all parameters listed.

Data Analysis

The methods of Kaplan and Meier[13] were used to produce the survival function, using the product-limit estimate model. Statistical significance was tested using the method described by Cox.[14] Hospitalization data were analyzed by analysis of variance.

Results

Figure 7 presents the survival of patients with low and high exercise capability who received low (NOT) versus high (COT) oxygen. Differences in survival between the low walkers on low oxygen and high walkers on high oxygen is statistically significant ($p = 0.01$). The difference is also significant between low walkers on low oxygen and low walkers on high oxygen ($p = 0.01$).

Figure 8 presents the hospitalization data in terms of the number of admissions per year and the average length of stay per admission for the 4 matched groups. Hospital utilization was less in the high oxygen group and least in the high oxygen group with "high" exercise capability ($p = 0.05$). Using analysis of variance, a statistically significant difference was found, with a shorter length of stay in the high walking subjects who received high oxygen ($p = 0.02$).

Discussion

The first long-term oxygen studies showed a reduction in pulmonary pressures and erythrocytosis in 6 patients in each of two studies with chronic stable hypoxemia associated with advanced COPD.[4,15] Somewhat similar studies of continuous oxygen from a stationary source also showed improvement in pulmonary artery pressure, with shorter duration of oxygen therapy, including as low as 15 hours per day.[16,17] These early observations formed the basis for both the British Medical Research Council and the NOTT randomized controlled clinical trials.

The British Medical Research Council study showed a survival benefit from oxygen given for approximately 15 hours per day, including during sleep, compared with air.[18] The NOTT study, which used ambulatory COT for as many hours per day as possible (median 19.4 h, mean 17.8 h/d), compared with NOT from a stationary source (mean 11.8 h/d), showed a better survival with more continuous oxygen.[9] Figure 9 shows a comparison of these survival curves.

Improved survival in the NOTT study was related to reversibility of pulmonary hypertension.[19] Similar conclusions have been drawn by other investigators.[20–22] It is difficult to understand, however, how a modest reduction in mean pulmonary artery pressure (only 3–5 mm Hg), which causes only a slight decrease in the afterload of the

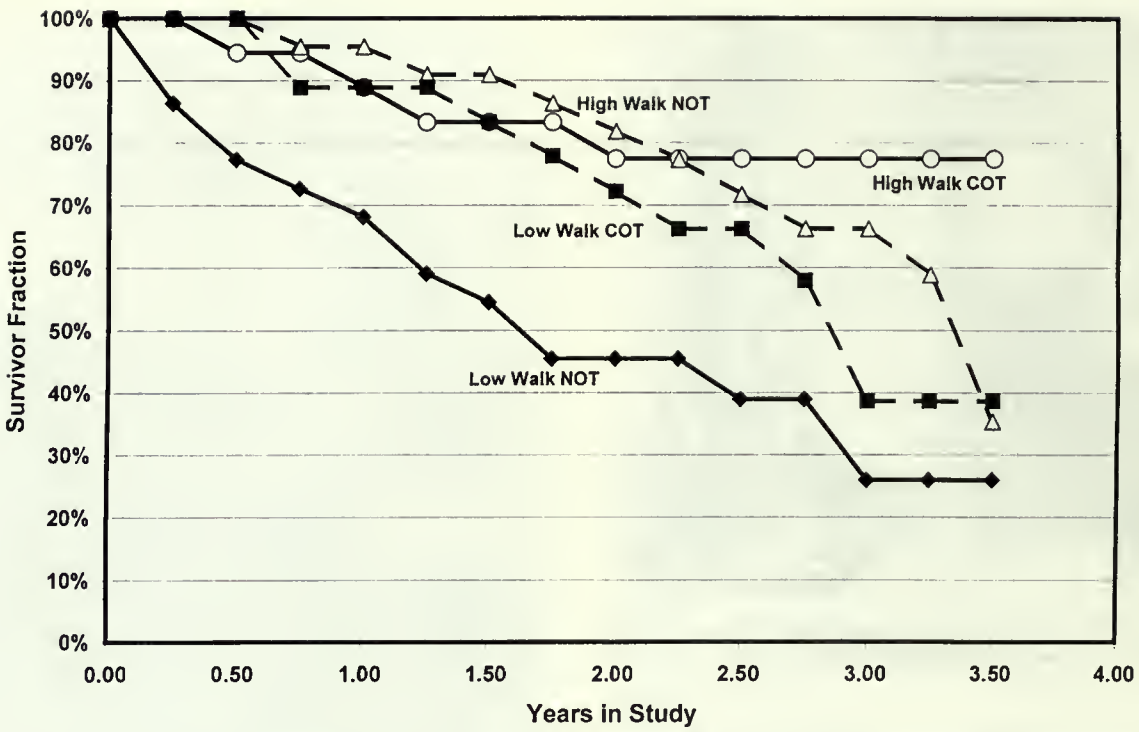

Fig. 7. Survivorship vs walking level and oxygen therapy. Comparison of survivorship in high walk, high oxygen patients compared with low walk, low oxygen patients shows highly statistically significant differences. Low walk, high oxygen survival is better than low walk, low oxygen at 2.5 years, but not at 3.5 years. These differences are statistically significant ($p = 0.01$).

right ventricle, could be the sole reason for the improved survival. In one study, improved survival was related to both a reduction in pulmonary pressure and an increase in maximum oxygen consumption.[20] In some patients who had a reduction in pulmonary artery pressure, there was an increase of left ventricular ejection fraction, which may be a reflection of improved global cardiac function because of relief of hypoxemia.[20]

Extensive exercise studies of right ventricular function during exercise in COPD have shown that an increase in exercise tolerance is related to an increase in right ventricular function and increased oxygen consumption.[23,24] Increased exercise capacity in hypoxemic COPD often results from oxygen administration and is related to an improvement in right ventricular function.[23–25] Increased right ventricular function and oxygen consumption may occur in spite of increases in pulmonary artery pressure and pulmonary vascular resistance.[23–25] The elevated pulmonary pressure and resistance are most likely due to fixed vascular changes in the pulmonary vascular bed, which are not reversible, even with long-term oxygen therapy (LTOT).[26] One study found a relationship between mixed venous oxygen tension and survival in patients who were candidates for LTOT.[27] The 5-year survival was better in patients with the highest mixed venous P_{O_2} and higher coefficient of oxygen extraction.[27] This study suggested that measures designed to increase cardiac output would be appropriate to improve tissue oxygen delivery.[27,28]

In other studies, patients who exhibited little or no ability to increase their cardiac output and systemic oxygen transport did not have increased exercise capacity.[23–25] These observations led to the conceptualization of the "right ventricular hypothesis."[29] The right ventricular hypothesis is based on the concept that limitation of right ventricular function limits systemic tissue organ transport. Before oxygen is given, ventricular function and oxygen transport may be limited by poor oxygenation of the myocardium. After oxygen is administered, right ventricular function can still be limited by structural damage of the right ventricle or by elevated right ventricular afterload from fixed pulmonary vascular changes. In any event, failure to improve right ventricular systolic function may limit tissue oxygen transport. By improving oxygen to the right ventricular myocardium, and by reducing right ventricular afterload, right ventricular function may improve. This, in turn, results in improved global cardiac function, increased tissue oxygen transport, and increased tissue energy production.

It is likely that tissue metabolic phenomena are responsible for improved survival. In the British Medical Research Council study, in men who received oxygen versus

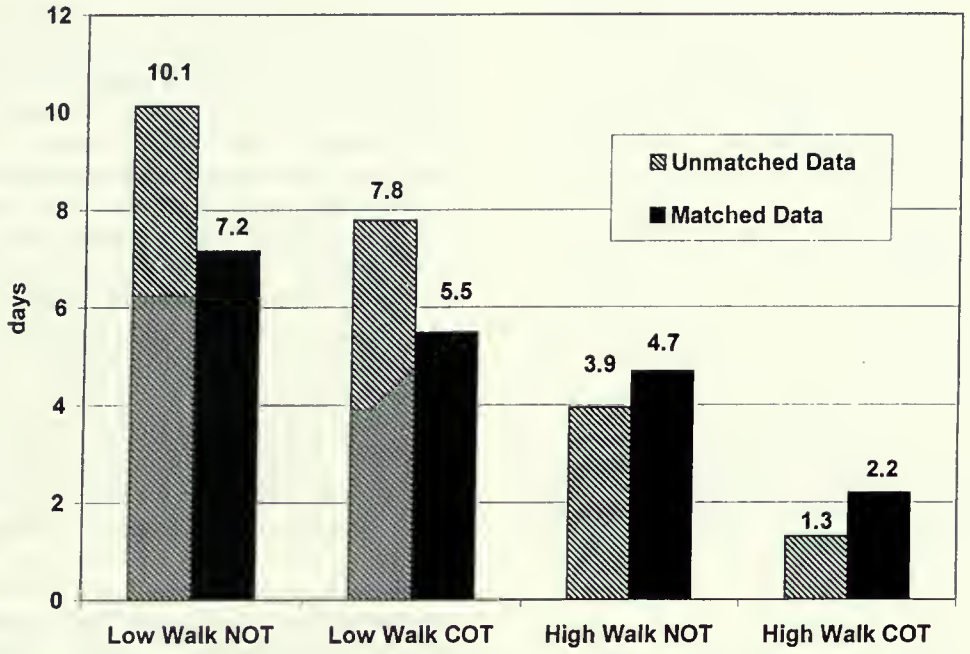

Fig. 8. Hospital days per year in study. The lowest length of stay in admissions per year was in the high walk, high oxygen patients. (p = 0.05 and 0.01 respectively). NOT = nocturnal oxygen therapy. COT = continuous oxygen therapy.

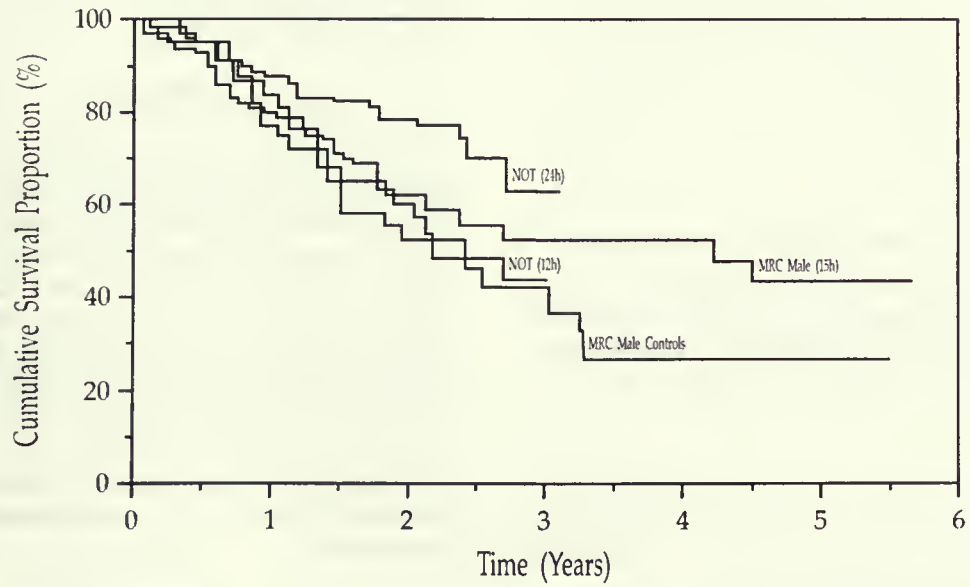

Fig. 9. Comparison of survival curves of Nocturnal Oxygen Therapy Trial (NOTT) and British Medical Research Council (MRC) study. (From Cooper CB, Long-term oxygen therapy. In: Casaburi R, Petty TL, editors. Principles and practice of pulmonary rehabilitation. Philadelphia: WB Saunders; 1993: p 184, with permission.)

men who did not receive oxygen, improved survival did not become apparent until after 500 days.[18] In the NOTT study, improved survival did not become statistically significant until 18 months (see Fig. 9).[9] These data suggest that the survival benefit from oxygen was a function of restorative metabolic changes in multiple organs, which occurred over months of LTOT.

The restorative value of oxygen in brain function has been previously reported.[30] Figure 10 shows an improvement in performance IQ, which was similar in both NOT

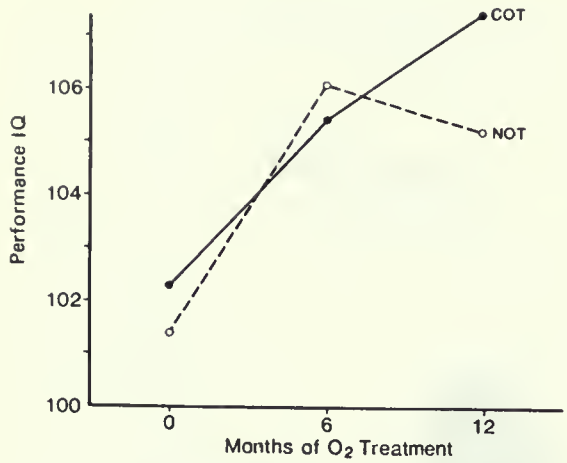

Fig. 10. Performance IQ from the Wechsler Adult Intelligence Scale in relation to duration of continuous (COT) versus nocturnal (NOT) oxygen therapy in COPD patients. (From Reference 30, with permission.)

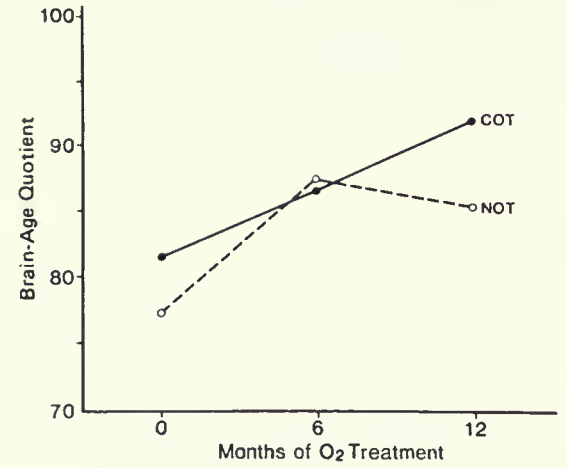

Fig. 11. Change in "fluid intelligence" (brain age quotient) in relation to continuous (COT) versus nocturnal (NOT) oxygen treatment in COPD patients. (From Reference 30, with permission.)

and COT. However, in COT there was further improvement over the subsequent 6 months. Similarly, Figure 11 shows improvement in brain age quotient, with improvement in both NOT and COT after 6 months, but further improvement after 12 months with COT.[30] An improvement in brain function could reasonably be expected to require increased energy production. Such an increase in energy production would require increased systemic oxygen delivery. This has been demonstrated in patients who can increase their exercise tolerance with supplemental oxygen.[22–24] We believe that increased exercise tolerance and improved survival are a result of increased systemic oxygen delivery during oxygen administration.

A restoration of arterial blood oxygenation while breathing air has been reported following several months of LTOT.[31] This improvement in oxygen transfer across the lungs is believed to be due to improved ventilation-perfusion matching as a result of improved oxygenation, which is known to reduce pulmonary arteriolar constriction and bronchospasm.[32] The long-term impact of LTOT on heart, brain, lung, and skeletal muscle function may also be due to a sustained increase in tissue oxygen transport and improved energy production of multiple organ systems.

A weakness of our study is that it is a retrospective analysis of a limited number of patients. But the NOTT study involved extremely well-characterized patients with exercise physiology and hospitalization outcomes. The present study suggests that improved tissue oxygen transport occurs in patients with relatively better exercise capacity who also receive more continuous ambulatory oxygen. Patients with lower levels of exercise capacity had better survival with oxygen delivered for longer periods of time.

This study suggests that new clinical trials are needed to compare outcomes from ambulatory oxygen for as many hours per day as possible, compared with stationary oxygen for an equivalent length of time. Such studies could answer the critical question about survival in relation to an ambulatory or stationary oxygen delivery source, and address the possibility of reducing hospitalizations with continuous ambulatory LTOT.

Summary

The NOTT study showed improved survival in COT patients who received LTOT for longer periods (mean 17.7 h/d, median 19.4 h/d) from an ambulatory oxygen system, compared with the survival of NOT patients who received oxygen for a mean of 11.8 h/d from a stationary system. The differences in survival could have been due to the method or the duration of oxygen therapy, or both. An increase in cardiac output and increased oxygenation of the arterial blood (oxygen content) results in increased tissue oxygen transport. In addition, COT was associated with better survival and reduced hospitalizations, compared with NOT patients who were unable to increase their walking level.

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Discussion

Kacmarek: I just wanted to ask you to comment on the restorative effects that you were discussing. You indicated that if a patient is on oxygen therapy because he or she is hypoxemic at rest, and he or she recovers from that a year or two down the road so that resting P_{O_2} is in the 60s, you would continue oxygen therapy. How about a patient who is discharged (after an acute exacerbation) with a P_{O_2} that necessitates oxygen therapy, but after a month or two they've recovered? Is that a patient who should have oxygen discontinued?

Petty: That's a very good question, and we addressed it during the oxygen consensus conferences. Certainly, with that unstable patient who has just come out of an exacerbation (often a pneumonia or just an exacerbation of COPD), we want them to go on home oxygen so they can get out of the hospital fast. We're not going to commit him or her to life-long oxygen, though, because he or she may not be a candidate in terms of being in a stable, steady state. I'm only recommending continued oxygen for patients who have a long-term, demonstrable need after a good stabilization period and then improved their ventilation-perfusion matching but don't change their overall basic spirometry. Those are the ones who shouldn't be taken off oxygen. And, Walter, you might want to elaborate on that because this was your concept.

O'Donohue: I agree with Tom that there are those patients who need short-term oxygen therapy until they recover from their acute illness, and they should not be subjected to a lifetime of oxygen therapy. The recommendation is that they should be retested in 1 to 3 months. But the patients that Tom is addressing are from 2 studies. One we did, and one was retrospective, looking at Weitzenblum's study.[1] These patients had been followed for some 40 months prior to beginning oxygen, and were shown to be hypoxemic with progressive pulmonary hypertension. They received oxygen for a year and then were restudied. At the end of one year of oxygen therapy, 25% of them would not have qualified for oxygen therapy. In our study,[2] on an average, patients had been on nasal oxygen for two years before starting transtracheal oxygen. So they had already been receiving oxygen, and at the time they started transtracheal oxygen, all of them had a P_{O_2} of $\leq$ 55 mm Hg. At the end of 6 months of transtracheal oxygen, 25% of them had a P_{aO_2} above 55 mm Hg. These were all stable patients, and this clearly appeared to be a reparative or a restorative effect of oxygen therapy and not simply the effect of instability of disease. But I would also mention that all of our patients who had $P_{O_2} > 55$ mm Hg desaturated with exercise. So they would have been experiencing periods of desaturation if we had stopped oxygen therapy. The assumption is that they would then revert back to more pulmonary hypertension and more of the disease that had caused the hypoxemia to begin with.

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Kacmarek: I agree totally with both of your responses, and the reason I ask was that many patients who get discharged from the hospital are not formally followed up the way they should be, and there is not that 1-month or 3-month follow-up to document whether oxygen therapy is still needed. Many of the patients who have been maintained relatively long-term on oxygen therapy at home are those who probably fit into that, but some of them fit into that group and may not need it. My concern was simply to get that recommendation back out on the table and into this document.

Pierson: What evidence is there that discharging a COPD patient who has a P_{O_2} of 50 following an exacerbation and not sending them home on oxygen adversely affects any outcome? I ask this because I suspect that in many countries patients who are admitted for an acute exacerbation are simply discharged on air, even if they remain hypoxemic after their several days in the hospital.

Criner: Your retrospective review based on low- and high-walk I found intriguing because this seems to be the first coupling of rehabilitation potential and prescription of LTOT. Do you believe that patients who are prescribed LTOT should be systematically examined in regard to their conditioning? If prescribed LTOT, should they also have reconditioning simultaneously applied? How do you interpret the data? Should everyone for whom we prescribe LTOT be assessed for their fitness and rehabilitation?

Petty: I think that would be ideal but impractical, because most oxygen in this country is prescribed by primary care physicians. Now maybe that would be very practical in Poland. But that's why I sort of plead for the definitive study, once and for all to find out in large groups of patients, although well-selected, like the NOTT study was, excluding about 80% of the patients, to get down to the ones that we really found as candidates, to answer the question so that industry knows what to do, so that payers know what to reimburse for. I think that's what really needs to be done.

Zieliński: I think that Poland is very well suited to do such a study, because in Poland the oxygen treatment is based only on oxygen concentrators. We cannot afford liquid oxygen, so without depriving the patients of any choice, we can randomly put some patients on ambulatory liquid oxygen systems and others on stationary oxygen. I will also show in my talk that we have very close follow-up of our patients. I think it would be a good idea to arrange such a study in Poland.

Wedzicha: I also found your high-walk vs low-walk data intriguing. There is one problem. We published a study where we performed a randomized, controlled trial of pulmonary rehabilitation in two groups of patients.[1] One group were the usual patients who are always studied, whom we called moderately disabled, though their forced expiratory volume in the first second measurements were about 37% of predicted. Our second group of patients was housebound, and these patients were mainly already on LTOT. Our rehabilitation experience was poor in that housebound group. We did not manage to improve exercise capacity, despite sending therapists to the patients' homes twice a week for 8 weeks. I think the limiting factor will be the ability of some of these patients to rehabilitate, for the reasons that Neil has already told us—they become deconditioned and they don't increase their heart rate response to exercise. Rehabilitation has to happen at a much earlier time in the natural history of COPD. What do you think of that, Tom?

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Petty: Well, if my hypothesis is correct, I agree that we should be doing pulmonary rehabilitation earlier on, and actually there are some data that speak to this. Rick Carter wrote a very interesting article about maximum oxygen consumption and maximum heart rate in people with only mild to moderate degrees of COPD,[1] on people in their mid-40s with forced expiratory volume in the first second measurements around 2.5, who aren't impaired by their ventilatory mechanics at all. But guess what? Their cardiovascular responses were terrible. They can't do a maximum heart rate, a maximum oxygen consumption, and they stop exercising, because they say they're tired. Well, I think the reason they're tired is they're having problems with oxygen utilization at the tissue level. We didn't go into the inflammatory cytokines that these patients have that are poisoning their mitochondria, too. We've got to interface the external respiration with internal respiration metabolism. I think the respiratory metabolic-interface is where the answer is.

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Oxygen and Air Travel

James K Stoller MD

Introduction
Effects of Acute Altitude Exposure
Conditions Inside Commercial Airplanes in Flight
Medical Hazards Associated with Commercial Air Flight
Prescribing Supplemental Oxygen for Commercial Air Flight
Practical Aspects of Flying with Supplemental Oxygen
Interaction with a Physician
Interacting with the Air Carrier and/or Travel Agent
Summary
[Respir Care 2000;45(2):214–221]*Key words: oxygen therapy, air travel, altitude exposure, in-flight oxygen.*

Introduction

Since the topic of commercial air travel for the technology-dependent individual was reviewed in RESPIRATORY CARE in 1994,[1] advances in this field have occurred, justifying an updated review. In this context, the current paper will present a comprehensive review of air travel with supplemental oxygen. As in earlier reviews,[1–4] the effects of acute altitude exposure are addressed first. Atmospheric conditions within commercial air carriers are then reviewed, followed by a discussion of the medical hazards associated with air travel. Strategies to predict in-flight arterial partial pressure of oxygen (P_{aO_2}) are discussed next, and, finally, recent information regarding airline policies about providing in-flight oxygen are summarized. In so doing, my purpose is to provide a useful review for the respiratory care clinician involved in evaluating and offering recommendations to prospective travelers in need of in-flight supplemental oxygen.

Effects of Acute Altitude Exposure

The physiologic effects of exposure to altitude are defined by two of the gas laws:[5] Dalton's law and Boyle's law. Dalton's law describes the relationship between partial pressure and total pressure of gases in a mixture, and states that the total pressure of a mixture of gases must equal the sum of the partial pressures of all of the component gases. Boyle's law describes the relationship between the pressure and the volume of a gas, such that the volume varies inversely with the gas pressure.

With regard to the impact of these gas laws during altitude exposure, the hypobaric conditions of altitude exposure cause the total barometric pressure to decrease, causing the partial pressure of each component gas to decrease in proportion to its percentage composition of the total. Stated simply, the partial pressure of oxygen (P_{O_2}) decreases with altitude. Similarly, as the ambient pressure falls during ascent to altitude, the volume of the gases in an enclosed space increases and gas density decreases. Table 1 presents measurements of atmosphere and tracheal P_{O_2} and of gas density as barometric pressure falls with ascent. As a general "rule of thumb," tracheal P_{O_2} declines 5 mm Hg per 1,000 feet ascended. Also, at an elevation of 8,000 feet, gas density is approximately 30% lower than at sea level.

With these changes accompanying ascent to higher altitude, many of the pulmonary effects of acute altitude exposure can be ascribed to the decrease in P_{O_2}. For example, as the ambient partial pressure of oxygen decreases, respiratory rate and minute ventilation increase, as does the ventilatory equivalent (minute ventilation/oxygen consumption). Similarly, altitude exposure produces hemodynamic effects, including accelerated heart rate, increased cardiac output, and redistribution of perfusion to essential

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Table 1. Changing Properties with High Altitude

Altitude (ft)	Barometric pressure (mm Hg)	Atmospheric P_{O_2} (mm Hg)	Tracheal P_{O_2} (mm Hg)	Gas density*	Gas volume
0 (sea level)	760	159	149	1.0	1.0
2,000	707	148	138	—	—
4,000	656	137	128	0.9	1.1
5,000	632	132	122	0.8	1.2
8,000	564	118	108	—	1.4
10,000	523	109	100	0.7	1.5
15,000	428	89	80	0.6	1.9
30,000	226	47	38	0.3	4.0
40,000	141	29	20	0.2	7.6

*Ratio of density at altitude to density at sea level.
P_{O_2} = partial pressure of oxygen.
(From Reference 6, with permission.)

organs, much as with the "diving reflex." Finally, neurologic sequelae of altitude exposure include cerebral vasodilatation and various neuropsychologic effects, such as inattention, sleepiness, headache, and memory impairment. Other effects of altitude exposure relate to gas expansion. For example, gas trapped in enclosed spaces, such as in sinuses, noncommunicating lung bullae, pleural spaces, or abdominal viscera may expand, causing such diverse clinical phenomena as flatulence, acute ear pain, expanding pneumothorax, or even actual dehiscence of abdominal wounds related to abdominal distention. In the era of prosthetic implants containing both air and liquid, the air within these prostheses may expand much like distended potato chip containers at altitude, causing unusual clinical events.[7]

Despite these protean effects of altitude exposure, relatively brief exposure (ie, < 12 h) to the altitude equivalent encountered during commercial air flight (ie, 8,000–10,000 feet, see below) seems to be extremely well-tolerated, even among patients with chronic obstructive pulmonary disease (COPD).[8,9] For example, in a study in which 8 COPD patients with mean forced expiratory volume in the first second (FEV_1) measurements of 42% of predicted were assessed during ascent to 1,920 m atop Mt Washington, Graham and Houston[8] observed early mild fatigue, with no significant changes in minute ventilation, respiratory rate, or resting heart rate. Similarly, when Berg et al[9] exposed 18 COPD patients (mean FEV_1 0.97, 31% of predicted) to 8,000 feet for 45 minutes inside a hypobaric chamber, no significant cardiopulmonary effects were observed, even as the mean P_{aO_2} fell to 47.4 mm Hg.

In summary, notwithstanding the many physiologic effects of altitude exposure, empirical studies of short-term altitude exposure to modest altitudes like those encountered during commercial air travel show good tolerance, even by travelers with advanced COPD.

Conditions Inside Commercial Airplanes in Flight

Although the concept that commercial airliners are "pressurized" is well-known, the results of a personal survey conducted while traveling suggest that details of the altitude equivalence in-flight are poorly understood, even by many airline employees. Although commercial airplanes may fly at altitudes up to 44,000 feet, the fuselage structure is built to withstand a transmural pressure gradient such that the internal pressure is maintained at the equivalent of 10,000 feet or less. Indeed, Federal Aviation Administration requirements dictate that the so-called "cabin altitude" (the altitude equivalent within the cabin to which passengers are exposed) be maintained below 8,000 feet under most travel circumstances.[1–3] Actual cabin altitudes have been measured in a variety of commercial airplane types by Cottrell.[10] As shown in Table 2, structural properties of different types of commercial airplanes permit different pressure gradients to be maintained. Put simply, the greater the tolerable differential pressure, the lower the cabin altitude during flight. As such, the Concorde, with a differential pressure of 10.7 psi can maintain a cabin altitude of 1,000 feet, compared with a Boeing 737, in which cabin altitude is 8,000 feet because of the lower differential pressure of 7.45 psi. Cottrell[10] found that more modern airplane types are capable of higher flight while exposing travelers to higher cabin altitudes, though still below the "ceiling" of 8,000 feet.

Medical Hazards Associated with Commercial Air Flight

Much interest has focused on the frequency and types of adverse medical events encountered during commercial air

Table 2. Commercial Aircraft Cabin Pressurization

Aircraft Type*	Differential Pressure (psi)	Cabin Altitude (ft)†
B-727	8.6	5,400
B-757	8.6	5,400
B-767	8.6	5,400
B-747	8.9	4,700
B-737	7.45	8,000
DC-8	8.77	5,000
DC-9	7.76	7,300
DC-10	8.6	5,400
A-300	8.25	6,100
A-320	8.3	6,000
L-1011	8.4	5,800
BAC-111	7.5	7,900
Concorde	10.7	1,000

*n = 204 flights on 28 commercial airlines with 16 different aircraft.
†Cabin altitude higher with new aircraft (mean 7,004 ft vs 5,820 ft, p = 0.002).
(Adapted from Reference 10, with permission.)

travel, for 3 important reasons: (1) exposure to hypobaric hypoxic conditions during commercial air flight poses some risk, albeit small; (2) individuals with medical conditions comprise an important minority of the flying public; and (3) commercial air carriers are sometimes used to transport patients with recent illnesses and/or end stage cardiopulmonary disease for further care (eg, transplantation).[11,12] As evidence of the frequency of commercial air flight by individuals with medical problems, Gong has estimated that 5% of air travelers are under medical care.[3] The acuity and types of medical illnesses among commercial air travelers was described by Gong et al in a survey of 1,115 passengers referred to Delta Airlines' medical evaluation service in 1991.[13] Thirty-five percent of these individuals reported being hospitalized within 2 weeks of arranging the air flight. Ten percent of these 1,115 individuals were deemed medically unfit for travel and were denied air passage. Of the 892 individuals for whom oxygen was recommended, the need was based on COPD in 44.3% and on cardiac disease in 34.1%. Finally, that travelers infrequently seek medical advice before travel was suggested by the results of a poll of 62 passengers with lung disease.[14] Thirty-two percent had taken a commercial air flight within the previous 2 years, but only 30% had sought medical advice before the actual trip. Also, of the 892 travelers using in-flight oxygen in the aforementioned series by Gong et al,[13] 72.8% had flown previously, but only 25.1% had previously used oxygen in flight.

In addressing a special subset of commercial air travelers with medical conditions, Cox et al[12] described 196 travelers who had experienced recent myocardial infarction. Specifically, the median time since myocardial infarction was 15 days, with a mean of 17 days and a range of 3 to 53 days. Ninety-five percent of these travelers experienced no in-flight symptoms or adverse outcomes. However, in 9 travelers (5%) symptoms occurred, attributed to a cardiac cause in 6 of the patients and to noncardiac causes in the remaining 3 (ie, gastroenteritis, urinary retention, and agitation). One individual who experienced gastroenteritis in flight died from a cardiac arrest and hypotension 8 hours after arrival, for an overall travel-associated mortality rate of 0.5%. Overall, this experience suggests that commercial air travel by patients experiencing recent myocardial infarction can be undertaken with relatively low risk. In a separate experience with 21 patients with severe cardiopulmonary disease being transported on commercial air flights to specialized centers (eg, for transplantation), Kramer et al[11] reported one death (of a mechanically ventilated patient with severe pulmonary hypertension from chronic emboli). In the remainder of the 20 patients, 6 had room air P_{aO_2} values that ranged from 40 to 59 mm Hg, but tolerated commercial air flights of 4 to 21 hours duration without adverse sequelae. Overall, given the large volume of commercial air travel currently and the frequency of travel by individuals with active medical problems, the low rate of adverse events is both reassuring and perhaps even surprising.

Against this background of frequent travel by passengers with medical conditions, the frequency of adverse medical events during commercial air travel is noteworthy.[15–20] Table 3 presents the findings of 4 available series that measured the overall frequency of medical emergencies during commercial air travel. The frequency of in-flight medical emergencies among travelers in series evaluating between 8.74 and 245.3 million travelers was similar, at 1 event per 19,000–39,600 travelers.[15–20] Death during commercial air travel was 100-fold less common, reported once among 3.2 million travelers in the series by Cummins et al.[15] The types of medical problems encountered during commercial air travel are summarized in Table 4. In many of the 6 available series,[15–20] cardiac events (eg, myocardial infarction, chest pain, heart failure) accounted for the most events, including 56% of deaths in one series.[15] Syncope and near syncope comprised 4–29% of events, gastrointestinal problems 11–12%, and pulmonary problems (eg, dyspnea, exacerbations of asthma and/or COPD) accounted for 6–12% of events.

Finally, given the surprisingly low event rates described among commercial air travelers, the possibility of at least two sources of bias must be considered: under-reporting of nonemergency adverse events by travelers, and denial of travel to severely medically-impaired travelers, as was reported in 10% of 1,115 travelers referred to the Delta Airlines medical evaluation unit.[13]

Table 3. Summary of Available Reports of Medical Emergencies during Air Travel

First Author (Year)	Site or Source	Number of Travelers	Frequency of Emergencies (*n*)	
			All Travelers	In Flight
Cummins (1988)[15]	IATA Survey	245.3 million	1/3,200,000 deaths	—
Cummins (1989)[16]	Sea-Tac	14.4 million (274,000 flights)	1/19,000 (754)*	1/39,600 (190)
Speizer (1989)[17]	Los Angeles International Airport	8.74 million	—	1/33,000 (260)
Skjenna (1991)[20]	Air Canada	13,353,000	3.4/100,000†	—

IATA = International Air Transport Association.
Sea-Tac = Seattle-Tacoma International Airport.
*Unscheduled landings in 7/190; 5 cardiac arrests of which only 1 occurred on board plane.
†Total 2 deaths.
(From Reference 1.)

Table 4. Types of Medical Problems Encountered during Air Travel: Summary of Available Series

First Author (Date)	*n* Events	Syncope/Near	Cardiac	Asthma/COPD	Seizures	Diabetes	GI
Cummins (1988)[15]	577 deaths	—	56%	6%	—	—	—
Cummins (1989)[16]	754	4%	11%	7%	4%	2%	12%
Speizer (1989)[17]	260	9%	17%	12%	5%	—	11%
Cottrell (1989)[18]	362	29%	19%	10%	4%	3%	—
Hordinsky (1989)[19]	1,016 events	23%	14%	2%	4%	2%	11%
	13 deaths		61%				
Skjenna (1991)[20]	464	—	51%	10%	7%	—	12%

COPD = chronic obstructive pulmonary disease. GI = gastrointestinal.
(From Reference 1.)

Prescribing Supplemental Oxygen for Commercial Air Flight

Because hypobaric hypoxia is believed to be responsible for the small risk of in-flight adverse medical events, supplemental oxygen has been recommended for travelers whose P_{aO_2} is predicted to be $\leq$ 50 mm Hg at cabin altitude.[1–4] Three general strategies have been proposed to predict the P_{aO_2} value at 8,000 feet of altitude: (1) actually exposing patients to hypobaric hypoxic conditions (eg, ascending to 8,000 feet in a hypobaric chamber,[21,22] by plane, or by mountain ascent);[8] (2) administering a hypoxic gas mixture to simulate the F_{IO_2} at 8,000 feet (ie, 15.1% oxygen);[23,24] and (3) using prediction equations or nomograms[21–26] derived from studies using hypoxic inhalation (ie, hypoxic hypoxia) or exposure to hypobaric conditions (ie, hypobaric hypoxia). Among these 3 strategies, actual measurement of P_{aO_2} under conditions of hypobaric hypoxia is used infrequently, being confined to settings where an altitude chamber is available for clinical testing. In a technique called the hypoxia altitude simulation test, hypoxic exposure to 8,000 feet equivalent is simulated by having the patient breathe 15.1% oxygen.[23,24] As an alternative method of creating an hypoxic gas mixture, Vohra and Klocke[27] proposed using a 35% Venti-mask through which high-flow nitrogen is administered. The gas mixture created with the entrained air is 16.1% oxygen, allowing simulation of a cabin altitude equivalent to approximately 6,700 feet. Advantages of this air-entrainment mask technique[27] include obviating the need for ordering and maintaining cylinders of 15.1% oxygen and providing the ability to titrate supplemental oxygen through a nasal cannula during the hypoxia altitude simulation test.

In clinical practice, regression equations are the most frequently used strategy to predict P_{aO_2}. Table 5 presents the regression equations from available series. Notably, earlier equations contain only the term "room air P_{aO_2} at ground level" as the independent variable,[23] while more recent equations contain both values of "room air P_{aO_2} at ground level" and "FEV_1"(or "FEV_1% predicted" or "FEV_1/forced vital capacity" as independent variables).[21,22,26] A recent meta-analysis of pooled data from 5 studies evaluating 71 COPD patients suggested yet other regression equations (see Table 5).[22]

For the clinician planning to use these equations, several important practical guidelines and resources are noteworthy. First, in a study of 22 COPD patients exposed to hypoxic hypoxia, Gong et al[4,23] found that a resting room

Table 5. Available Equations for Predicting In-Flight Pa_{O_2}

Equation		r
1	P_{aO_2} altitude = 0.84 + (0.68) P_{aO_2} ground	0.94
2	P_{aO_2} altitude = 0.453 (P_{aO_2} ground) + 0.386 (FEV_1 % pred) + 2.440	0.85
3	P_{aO_2} altitude = $[P_{aO_2}] \times e^{[-0.01731 - (0.00019)(FEV_1\ \%\ pred)](P_{IO_2}\ ground - P_{IO_2}\ altitude)}$	0.92
4	P_{aO_2} altitude = 0.294 [P_{aO_2} SL] + 0.086 [FEV_1 % pred] + 23.211	0.804
5	P_{aO_2} altitude = 0.245 [P_{aO_2} SL] + 0.171 [FEV_1/FVC % pred] + 21.028	0.828
6	P_{aO_2} altitude = 0.238 [P_{aO_2} SL] + 20.098 [FEV_1/FVC] + 22.258	0.827

r = correlation coefficient.
P_{aO_2} = arterial partial pressure of oxygen.
SL = sea level.
FEV_1 % pred = percent of predicted forced expiratory volume in the first second.
(Modified from References 21, 22, and 26.)

air $P_{aO_2} < 72$ mm Hg predicted an in-flight $P_{aO_2} \leq 50$ mm Hg in 86.7% of subjects. Conversely, a sea level $P_{aO_2} > 72$ mm Hg makes an in-flight $P_{aO_2} < 50$ mm Hg unlikely. Second, computer-based interactive programs are available that make calculation of in-flight P_{aO_2} easy.[27,28] While these regression equations and "rules-of-thumb" are helpful, several cautions about their use are also noteworthy. First, the equations require validation in data sets other than those used to derive the equations. Second, use of these equations should be restricted to the types of patients included in the derivation studies. Thus, currently available regression equations do not apply to patients with COPD who also have co-morbid conditions (eg, anemia, cardiovascular disease, stroke), hypercapnic COPD, or restrictive lung disease.[21–25] Patients with such conditions, for whom in-flight oxygen is being considered, should undergo testing with hypoxic gas mixtures in order to determine whether in-flight P_{aO_2} would fall below 50 mm Hg.

Once it has been determined that the patient's in-flight P_{aO_2} would be < 50 mm Hg and supplemental oxygen is deemed necessary, the requisite liter flow of supplemental oxygen must be determined. In addressing this question, Cramer et al[29] studied 20 patients with lung disease (10 with obstructive, 10 with restrictive) needing supplemental in-flight oxygen, and reported that 2 L/min sufficed to maintain arterial oxygen saturation (as measured via pulse oximetry) at ≥ 90% in 95% of subjects. In only a single study subject with severe obstructive lung disease (ie, FEV_1/forced vital capacity = 0.36, diffusing capacity of carbon monoxide = 36% predicted) was a higher liter flow (ie, 3 L/min) required.

Finally, for patients using long-term supplemental oxygen at home who plan commercial air travel, no specific guidelines exist for prescribing in-flight oxygen. However, extrapolating the results of Cramer et al[29] suggests that a 2 L/min increase in flow will generally suffice to assure adequate P_{aO_2} during flight.[11] If a simulation is desired to determine the needed liter flow, the hypoxic inhalation test described by Vohra and Klocke[27] can be used while supplemental oxygen is bled in at increasing flows until an adequate saturation level is achieved.

Practical Aspects of Flying with Supplemental Oxygen

As reviewed previously[1,2] and presented in Table 6, practical aspects of planning a trip with in-flight supplemental oxygen can be considered in 4 phases: (1) interaction with a physician, (2) interaction with a commercial air carrier and/or travel agent, (3) personal planning for the travel day, and (4) interaction with an oxygen vendor (if supplemental oxygen is required upon arrival). New information regarding Phases 1 and 2 justify discussion here. Practical details regarding Phases 3 and 4 are described in Table 6, which is derived from an earlier review in RESPIRATORY CARE.[1]

Interaction with a Physician

Interaction with a physician is a critical aspect of planning a commercial air flight with supplemental oxygen, because medical evaluation is required to ascertain whether supplemental oxygen is needed, and, if so, to communicate this need to the commercial air carrier. Indeed, most commercial air carriers require a letter from a physician stating the traveler's medical condition for which supplemental oxygen is needed,[1,30] and both the liter flow and duration of in-flight oxygen needed. As an alternative to the physician's letter, the International Air Transport Association issues a Medical Information Form, which is required by some air carriers. Also, some carriers' medical departments issue a Frequent Traveler's Medical Card, which states the traveler's fitness to fly and obviates the need for a physician letter with each flight, but requires periodic review. It is also advisable for physicians to provide backup copies of prescriptions, an emergency supply of medications (eg, corticosteroids or antibiotics in the event of a

Table 6. Practical Tips for Planning Travel for the COPD Patient

Interaction with physician
· Determine whether need for in-flight oxygen exists
· Get multiple copies of:
-letter describing condition
-liter flow and duration of in-flight oxygen
-medication prescriptions
· Consider emergency supply of antibiotics, corticosteroids
Interaction with air carrier/travel agent
· Notify of need for oxygen ≥ 48 hours before flight
· Fly nonstop if possible:
-less inconvenient
-less expensive (especially if oxygen cost is per coupon)
· Travel during business hours so vendor personnel are available
· Pre-arrange motorized cart or wheelchair if layover is scheduled
· Try to be seated near bathroom on plane
· Call airline at least 48 hours before flight to confirm
· Consider using a travel agent specializing in travel for patients with medical needs
Personal planning for travel day
· Arrive at least 1½ hours early:
-Must pay for oxygen on day of travel
-Charge structure may vary (eg, either per coupon or per container)
· Bring your own nasal cannula and extra length of tubing
· Pack medications in carry-on luggage
· Have multiple copies of prescriptions
· If oxygen is needed for layover and not already arranged, first aid stations may have (35% have oxygen available, by informal poll)
· Have cash (to pay for oxygen if needed)
Interaction with oxygen vendor
· Favor a company that has or can arrange nationwide coverage
· Arrange for oxygen during layovers (if needed)
· Try to learn the type of system that will be supplied (ie, to check adapters)

COPD = chronic obstructive pulmonary disease.
(Modified from Reference 1.)

COPD exacerbation), and names of physicians at stopover and final destinations in the event that medical attention is needed.

Interacting with the Air Carrier and/or Travel Agent

In the context that standard regulations dictating the provision of in-flight oxygen are not available, there is great variation between carriers regarding whether in-flight oxygen is provided and, if so, the liter flow and interface options available. Also, the cost of in-flight supplemental oxygen varies widely among commercial air carriers. Importantly, the cost of in-flight supplemental oxygen is borne by the traveler, even if the traveler is eligible for Medicare reimbursement for long-term supplemental oxygen at home. Given the variation among carriers, general advice for the traveler is to call well in advance of the trip (ie, ideally weeks before) and to compare services and prices among air carriers serving the intended destination.

As evidence of the significant variation in oxygen policies and services among commercial air carriers, findings from a recently-conducted telephone survey of 33 commercial air carriers listed in the Cleveland, Ohio, Metropolitan Yellow Pages are summarized in Table 7.[30] Oxygen was offered by 25 of the 33 carriers sampled (76%) but was not offered by 8. In calls made by respiratory therapists posing as prospective travelers trying to arrange in-flight oxygen, the mean required time on the telephone was 9.96 minutes (range 3–20 min) with placing up to two calls and speaking with up to two airline representatives. Up to one month of advance notice to the carrier was required, all carriers required a physician letter, and both the types of oxygen equipment provided and the cost to the traveler varied greatly (ie, from no charge up to $1,500 for a "standard" trip of 6 hours total duration using a flow of 2 L/min). Because airline policies regarding supplemental oxygen may change over time, the traveler is advised to use the data in Table 7 as a general guideline, but to obtain updated information by directed inquiry. In addition to several printed resources,[30–32] a web site (www.oxygen4travelers.com) has been promoted by the American Association for Respiratory Care as a source of current information for oxygen-using travelers.

Finally, variability in airline policy and the resultant difficulty of arranging in-flight oxygen for travelers has spurred efforts by official organizations to effect policy reform. Indeed, efforts to standardize airline policy and costs and to enable travelers to bring Federal Aviation Administration-approved personal portable oxygen supply systems aboard commercial aircraft have been undertaken by the American Association for Respiratory Care, the American Medical Association,[33] and the National Association for Medical Direction of Respiratory Care.

Summary

In summary, the environmental stresses of commercial air travel and the possibility of altitude-induced adverse events during flight suggest the importance of evaluating prospective air travelers with chronic pulmonary disease for in-flight supplemental oxygen needs. Although gaps exist in current understanding of the risks of air travel and optimal ways of predicting the need for in-flight oxygen, respiratory clinicians can use available techniques to assess travelers' in-flight oxygen needs in many instances. Significant current variation among commercial air carriers in policies regarding providing oxygen, related equipment, and the cost of in-flight oxygen poses challenges to the prospective air traveler and warrants attention.

Table 7. Summary of Features Regarding Arranging In-Flight Oxygen for 33 Commercial Carriers Sampled

Carrier	Permits O_2?	Total Telephone Time (min)	Number of Calls Required	Number of People Spoken To	Advance Notice Required	MD Notification Needed?	Charge for a "Standard Trip"*	Devices Available	Liter Flow Options	Rating Value†
International										
KLM Royal Dutch	Yes	10	1	1	1 wk	Yes	0	simple mask, NC	2 or 10	3
China Airlines	Yes	15	2	2	1 wk	Yes	0	NC	1–6	2
Emirates	Yes	10	1	2	72 h	Yes	0	NC	2	3
Air Afrique	Yes	10	1	2	1 wk	Yes	$64	NC	2–4	2
BWIA	Yes	8	1	1	1 wk	Yes	$80	NC	2, 4, 6	2
Qantas	Yes	5	2	2	72 h	Yes	$100	venti-mask, simple mask, NC	1–15	1
Air Ontario	Yes	8	1	1	48 h	Yes	$120	NC	1 or 4	3
British Air	Yes	15	1	2	48 h	Yes	$150	NC	2 or 4	4
Lufthansa	Yes	20	1	2	72 h	Yes	$150	NC	2–8	2
Japan	Yes	6	1	1	1 mo	Yes	$250	NC	2	4
Canadian International	Yes	9	2	2	48 h	Yes	$260	NC	2 or 4	4
Mexicana	Yes	8	1	1	72 h	Yes	0	NC	Any	3
Swiss Air	Yes	5	1	1	1 wk	Yes	$274	NC	2–6	4
Singapore	Yes	4	1	1	2 wk	Yes	$1,100	NC	2–4	4
Alitalia	Yes	12	1	2	72 h	Yes	$1,500	NC	6	4
Aerolineas	No	1	1	—	—	—	—	—	—	5
Finn Air	No	1	1	1	—	—	—	—	—	5
Korean	No	3	1	1	—	—	—	—	—	5
Phillippine	No	2	1	1	—	—	—	—	—	5
Saudi Arabian	Yes	6	2	3	72 h	Yes	0	NC	2–4	2
Scandinavian	Yes	5	1	1	3–4 wk	Yes	0	NC	2–4	4
South African	Yes	6	2	2	3–4 wk	Yes	0	NC	2–4	4
United States-based										
America West	No	10	2	2						5
Delta	Yes	18	2	2	72 h	Yes	$100	NC	2–8	2
Midwest Express	No	5	1	1						5
United	Yes	15	2	2	48 h	Yes	$100	NC	2, 4, 6	3
American	Yes	10	2	2	48 h	Yes	$150	NC	2 or 4	4
TWA	Yes	7	2	2	48 h	Yes	$150	NC	2–8	4
Continental	Yes	16	1	2	48 h	Yes	$200	NC	2–4	3
US Air‡	Yes	10	2	2	72 h	Yes	$120	simple mask, NC	2, 4, 6	2
Northwest	Yes	15	1	2	48 h	Yes	$150	simple mask, NC	2–8	3
Southwest	No	3	1	1	—		—	—	—	5
Skyway	No	3	1	1	—		—	—	—	5

*Based on 1997 United States dollars.
†See text for explanation.
‡Now US Airways.
NC = nasal cannula.
(From Reference 30, with permission.)

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Discussion

MacIntyre: Jamie, this is just a technical kind of issue. When the airlines supply a tank, do they bolt it to the seat, or do you have to buy an extra seat? A seat belt? How does it work?

Stoller: That is variable, but in general, they have tanks that have been pressure-tested. They secure them in an under-seat carriage. Lufthansa does this by oxygen concentrators under the seat, which is the same strategy as used for military air transport. Obviously, this can technically be done, and we've seen examples in various talks during this meeting in which concentrators can be provided. So there are other strategies, but in general it's by pressurized gas in an undercarriage secured to the seat.

MacIntyre: I got my start in pulmonary physiology from being a flight surgeon. My first job was to study the pulmonary function effects of a molecular sieve attached to the back of the jet engine that functioned as an oxygen concentrator for military pilots. We found it actually worked very well. The gas also contained 4% argon. Although people were worried about that, the small amount of inert gas actually helped; it prevented atelectasis during high-G-force maneuvers.

Benditt: I find it easier to set up home mechanical ventilation for patients than to do this.Wasn't there a trial looking at the outcomes? Obviously,

there are a lot of data out there, but does it really affect the outcome?

Stoller: That's exactly the right question. There is reasonable concordance among most of the official societies that have commented on the need for in-flight oxygen and about the criteria for predicting in-flight P_{aO_2}. But there is precious little information that clearly shows increased risk to these individuals. So it's a murky area.

O'Donohue: Representing the National Association for Medical Direction of Respiratory Care, we met in Washington, DC, a few years ago with the Department of Transportation, the Federal Aviation Administration, and representatives from almost all the airlines, as well as industry. Everybody agreed with the concept, whereby when oxygen was prescribed the patient could pick up a sealed system for oxygen delivery (probably one with a conserving device) that they could carry from the beginning of their flight to the end. This would allow patients to use oxygen between flights when there may be 3–4-hour delays in airports. Everyone agreed that it could be done. The airlines thought it was a great idea. The industry said they had no problem shipping the devices back and forth. Then, soon after this meeting, the ValueJet accident occurred, and everything stopped, unfortunately.

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The Demographics and Economics of Long-Term Oxygen Therapy

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[Respir Care 2000;45(2):223–228] *Key words: long-term oxygen therapy, demographics, economics, compliance, Medicare, reimbursement, reassessment, incidence.*

Introduction

When appropriately prescribed and correctly used, long-term oxygen therapy (LTOT) represents an important clinical tool for the effective management of chronic hypoxemia secondary to chronic obstructive pulmonary disease (COPD).[1,2] LTOT improves survival and quality of life of COPD patients, and does so in a highly favorable cost-benefit ratio.[3] However, the benefits of home oxygen therapy are not realized without a steep price. For example, in the United States, the Health Care Financing Administration (HCFA) recently reported that total Medicare expenditures for home oxygen equipment in 1997 and 1998 were $1.7 billion and $1.3 billion, respectively, with the lower figure for 1998 a direct result of the implementation of drastic cuts in Medicare reimbursement for home oxygen equipment mandated in the Balanced Budget Act of 1997.[4] As a percentage of total Medicare expenditures for durable medical equipment, LTOT represented 36% in 1997, decreased to 28% in 1998, again as a direct result of mandated cuts in the monthly reimbursement rate. Table 1 summarizes the latter figures.

In 1997, home medical and respiratory therapy (HME/RT) equipment providers were paid an average of $300 per month to provide home oxygen therapy, regardless of whether the stationary equipment was an oxygen concentrator or liquid oxygen system. This indeed represents a very expensive undertaking. At this writing, because of Medicare cutbacks, the monthly payment is now in the $240 range, and even with this 30% reduction there are still those who continue to question the overall cost-effectiveness and affordability of LTOT. Ironically, the relentless focus on the size of the annual expenditure for home oxygen therapy, especially that portion paid by Medicare, clouds and sometimes all but obfuscates the cost-savings being realized by reduced utilization of acute and emergency care services by COPD patients whose chronic hypoxemia is effectively managed with home oxygen therapy.[5] The apparent disconnect between recognizing the cost-savings realized through reduced hospitalization at the expense of paying for LTOT is no doubt the result of having Medicare payments for acute hospital care and for home oxygen equipment provided by two separate parts of the Medicare Program, Part A and Part B. With COPD firmly entrenched as the fourth leading cause of death in the United States, claiming 107,170 deaths in 1997, and with more than 95,000 new, first-time patients being diagnosed with COPD each year, the debate over the cost-benefit of home oxygen therapy is not likely to be resolved any time soon.[6]

Number of Long-Term Oxygen Therapy Patients in the United States

Regrettably, an exact figure for the actual number of individuals in the United States currently receiving home oxygen therapy continues to remain elusive. Previous estimates ranged from a high of 800,000 individuals to a low of 537,225, with additional uncertainty from the fact that each estimate was for a different calendar year. The 800,000 number was originally suggested in 1988 by O'Donohue

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Table 1. Total Medicare Payments for DME and LTOT*

	1997	1998
Total DME	$4.7 billion	$4.5 billion
Total LTOT	$1.7 billion	$1.3 billion
LTOT % of DME	36%	28%

*Source: Health Care Financing Administration, Office of Information Services.[4]
DME = durable medical equipment; LTOT = long-term oxygen therapy.

and was extrapolated from data provided by the Congressional Budget Office and a few large home care companies.[7] However, O'Donohue and Plummer later revised this estimate, using data provided by the HCFA regarding the number of claims paid for home oxygen equipment during 1993.[8] Through their analysis of these claims data, O'Donohue and Plummer estimated that there were 394,780 Medicare patients using oxygen concentrators in 1993. Further, because 80% of Medicare patients were found to receive their home oxygen from concentrators during that period, the total Medicare population for all home oxygen was calculated to be 493,500.[9] Finally, it is also assumed that Medicare covers up to 80% of all patients in the United States using home oxygen therapy. Thus, the total estimated number of home oxygen patients in 1993 was 616,000.

A more recent attempt to estimate the population of LTOT users was reported by Silverman et al.[10] Using the HCFA National Claim History System 5% Physician Supplier File for 1991 and 1992, Silverman et al found that 21,489 Medicare beneficiaries in the sample had claims filed for home oxygen equipment in 1991. This 5% figure, when projected to the population of Medicare beneficiaries, yielded an estimate of 429,780 Medicare LTOT users in 1991. Applying the aforementioned 80% rule, this would in turn suggest 537,225 total home oxygen users nationwide.

In an effort to determine if either the O'Donohue/Plummer estimate or the Silverman estimate could serve as a barometer for more contemporary estimates, the author used recently-reported Medicare expenditure data and attempted calculations from a different approach. As mentioned, Medicare reported total expenditures for all stationary home oxygen equipment (concentrators and liquid systems) in 1997 to be $1.7 billion (Table 2). If it is assumed the average monthly Medicare payment in 1997 for home oxygen was $260 (80% of the 1997 monthly allowable of $328), an annual cost per beneficiary of $3,120 is calculated. Dividing the annual estimated cost per Medicare beneficiary into the total Medicare expenditures for home oxygen therapy equipment, we find the total number of Medicare patients to be 570,206. Using the 80% rule, we then calculate the total number of users of home oxygen in 1997 to be 712,758.

In 1998, total annual Medicare expenditures for all stationary home oxygen equipment were $1.3 billion and

Table 2. Medicare Expenditures for Home Oxygen Equipment, 1997–1998*

Equipment	1997	1998
Oxygen concentrator < 2 L/min	398,231,135	310,074,491
Oxygen concentrator 2–3 L/min	359,693,760	275,819,498
Oxygen concentrator 3–4 L/min	192,216,297	141,706,739
Oxygen concentrator 4–5 L/min	517,398,440	455,917,433
Oxygen concentrator > 5 L/min	94,543,316	65,045,933
Stationary liquid system	216,971,393	146,252,134
Total all stationary	1,779,044,341	1,394,816,228

*Source: Health Care Financing Administration, Office of Information Services.[4]
LTOT = long-term oxygen therapy.

reflected the 25% cut mandated by the Balanced Budget Act of 1997. The average monthly Medicare payment in 1998 was $196 ($2,352 annually). Using the same formula, we now calculate the total number of Medicare patients receiving LTOT in 1998 to be 593,034 and the total patient population to be 741,292. Table 3 summarizes the numbers for the 4 different estimations.

Missing from the above estimates is the fact that during each year a percentage of LTOT users stop using home oxygen, primarily as a result of death. No doubt a significant number of the 107,000 reported annual deaths in 1997 from COPD were LTOT users. However, when Silverman et al compared 1991 data with 1992, they estimated that there were approximately 128,000 new Medicare LTOT users in that year.[10] Based on those data, it is likely that there is a slight net increase in the total number of LTOT users in the United States each year, which would further support the more recent estimate of approximately 700,000.

Table 3. Estimates of Number of LTOT Users in the United States

Author	Silverman[10]	O'Donohue Plummer[8, 9]	Dunne	Dunne
Year	1991	1993	1997	1998
Total Medicare	429,780	493,500	570,206	593,034
Total all LTOT	537,225	616,000	712,758	741,292

LTOT = long-term oxygen therapy.

The differences between Silverman's estimate of 537,225 LTOT users in 1991, O'Donohue and Plummer's estimate of 616,000 users in 1993, and the proposed 1998 estimate of 741,292 users do point out the difficulty of attempting to "back into" an actual number of LTOT users in the United States. However, it is probably safe to assume that

these numbers reflect a reasonable range and are appropriate for a discussion of the economic impact of this important yet expensive therapy.

Incidence of Long-Term Oxygen Therapy Usage

At more than $1.6 billion per year, annual expenditures for home oxygen therapy in the United States far exceed what is spent in other countries. Further, the incidence of usage in the United States is likewise well beyond what is found in other countries. For example, the reported incidence in France is 26 per 100,000; in England it is 20 per 100,000; in Japan it is 19 per 100,000, and in Canada it is 60 per 100,000.[11] However, in the United States the incidence of usage is a staggering 241 per 100,000, with more patients having received LTOT in the United States than in all other countries combined.[3] This gives rise to speculation about the cause for such a high degree of utilization. It could well be that there simply is a much higher per capita incidence of chronic hypoxemia in the United States. Possibly some of the home oxygen therapy that is prescribed is medically unnecessary, but given the current restrictive coverage and reimbursement guidelines for LTOT, it is unlikely that much home oxygen is being prescribed in an inappropriate manner. Coverage and reimbursement criteria are now firmly established and include very specific laboratory documentation of the need for supplemental oxygen through the direct measurement of arterial oxygen tension or oxyhemoglobin saturation.[12,13] Although these guidelines for coverage and payment, which are very similar to criteria followed in the Nocturnal Oxygen Therapy Trial (NOTT), were originally established for the Medicare program, they are now almost universally used by Medicaid, the Department of Veterans Affairs (VA), and most commercial health plans. It is therefore unlikely that there is a significant volume of home oxygen therapy inappropriately prescribed, because payment would not be forthcoming without sufficient proof that established coverage criteria are satisfied.

In an attempt to explain the high LTOT usage in the United States, O'Donohue and Plummer offered the following reasons.[8] First, arterial blood gas analysis and oxyhemoglobin saturation measurements are more commonly obtained in patients with COPD. Second, patients for LTOT are being identified earlier. Third, there is a greater awareness of the benefits of LTOT on the part of physicians and other health care providers. Fourth, there is a wide availability of HME/RT equipment providers eager to supply oxygen equipment. Fifth, reimbursement is readily available for most patients who satisfy coverage criteria for LTOT.

O'Donohue and Plummer are probably correct in the reasons they put forth to help explain the inordinately high usage of LTOT in the United States. However, one could also argue that major impetus for high LTOT usage has more to do with the relentless sales and marketing efforts of the various home care companies who have historically realized a major percentage of their annual revenues from home oxygen reimbursement. With more than 108,000 Medicare Supplier Numbers issued nationwide, there is certainly no shortage of HME/RT providers.[14] It is important to realize that most, if not all, HME/RT providers, whether they be small privately owned, or large publicly-traded companies, are structured and operate as for-profit entities. This is in sharp contrast to Medicare-certified home health agencies, many of which are established as not-for-profit entities. For-profit companies, especially large national or regional HME/RT providers, are driven by the recurrent need to increase annual revenues and profitability, and for-profit home care companies deliver more services than do their not-for-profit counterparts.[15] Further, physician ownership of home care companies has likewise been shown to result in higher utilization,[16] although Medicare largely banned physician ownership of HME/RT companies in 1995.

Historically, Medicare oxygen payments could account for as much as 60–70% of the total revenues of an HME/RT provider. Accordingly, HME/RT companies have tended to focus on product lines believed to have good profit margins. Oxygen concentrators, which minimize resupply, distribution, and maintenance costs, have been highly prized in this regard. It is little wonder then that many of the larger HME/RT providers have very lucrative compensation packages for their sales associates based on the number of new Medicare oxygen referrals obtained each month. The increased awareness of the benefits of LTOT on the part of physicians, especially nonpulmonologists, is thus more likely attributable to the tenacity of a highly motivated and skilled sales force than to any other source.

Another potential explanation for the unusually high number of LTOT patients in the United States may be the manner in which Medicare reimburses for home oxygen equipment. Once a Medicare beneficiary qualifies for LTOT coverage by showing a resting, room air, arterial oxygen tension $\leq$ 55 mm Hg or oxyhemoglobin saturation $\leq$ 88%, and the required documentation is accurately completed (HCFA Form 494 – Certificate of Medical Necessity: Oxygen), monthly payments for the rental of the oxygen equipment are nothing short of automatic. In fact, with the advent of electronic claim submission it is literally through several strikes of a computer keyboard that claims are forwarded each month to the Medicare carrier for processing. With over 80% of all claims for LTOT submitted electronically, this ease of obtaining reimbursement cannot be discounted (Health Care Financing Administration, Office of Financial Management, Baltimore, MD. May 1999, personal communication). There are virtually no requirements for subsequent reassessment of the ongoing

medical need for LTOT nor any requirement that continued compliance with the treating physician's prescription be verified as a condition of continuing coverage. This is especially troublesome because there is now ample evidence that continued compliance with the original prescription for LTOT is not uniformly assured.

Are Long-Term Oxygen Therapy Patients Compliant?

In spite of the abundance of scientifically validated studies demonstrating the positive impact of LTOT on survival and quality of life for chronically hypoxemic COPD patients, there is a growing concern about the overall degree of sustained adherence to the treating physician's prescription. Though no studies have been reported in the United States, several have been carried out in other countries. In the United Kingdom, Walshaw et al studied 67 patients receiving LTOT via oxygen concentrator, and found that only 55% were using oxygen therapy correctly.[17] Also in the United Kingdom, Howard et al evaluated 531 LTOT patients who were prescribed oxygen concentrators, and reported that 50% of the patients were not using the equipment at the level at which clinical benefit could be expected.[18] Pepin et al studied 930 LTOT users in France, and observed that only 45% achieved therapy for 15 hours or more per day.[19] In a Spanish study of 62 LTOT users, Granados et al reported an astounding 69% noncompliance rate, although these researchers also reported that only 36 of the 62 users (58%) had what was identified as an "appropriate prescription."[20] Shiner et al evaluated 63 LTOT users in Israel, and found that only 33% used LTOT in the 12–24 hours per day range.[21] Finally, Farrero et al studied 128 LTOT patients in Spain, and found that LTOT use was appropriate in only 26%.[22] Table 4 summarizes the results of these studies.

Several reasons for such poor compliance have been proffered. Pepin et al suggest that a primary contributing factor is the failure of the prescribing physician to adequately inform the patient beforehand of the expected usage.[19] Kampelmacher et al evaluated surveys received from 528 LTOT users in the Netherlands, and reported that the most common complaint contributing to noncompliance was restricted autonomy.[23] Along the same lines, Ring and Danielson expressed concern over the social isolation they witnessed during their study of a relatively small number of LTOT users in Sweden.[24] Weitzenblum, commenting on the work of Pepin, believes that LTOT is often poorly prescribed and therefore provided inappropriately.[25] On the other hand, Farrero et al argue that a lack of follow-up through structured home visits and ambulatory care is the contributing factor.[22] The issue of LTOT noncompliance represents a very serious challenge, especially in the United States, where efforts abound to further reduce the monthly Medicare reimbursement for LTOT equipment.

Table 4. Results of Adherence/Compliance Studies for LTOT

Year	Researcher	Country	Users (*n*)	Rate (%)
1988	Walshaw[17]	United Kingdom	67	55
1992	Howard[18]	United Kingdom	531	50
1996	Pepin[19]	France	930	45
1997	Granados[20]	Spain	62	31
1997	Shiner[21]	Israel	63	33
1998	Farrero[22]	Spain	128	26

LTOT = long-term oxygen therapy.

Optimizing Long-Term Oxygen Therapy Usage

The continued willingness of health plans, including Medicare, to fund LTOT will depend on several issues, foremost of which will be evidence that the therapy is being used appropriately and that the results are medically justifiable. Appropriate use means that LTOT is prescribed for use as close to 24 hours per day, 7 days per week, as possible, and that the patient receives a clear and concise message from the physician on the importance of adhering to this prescription from the time therapy is started. HME/RT providers must reinforce this message at each opportunity, starting with the education and training that accompanies the initial delivery and setup of the oxygen equipment. Further, HME/RT providers must assume a more proactive role in monitoring continued patient compliance, and work closely with the prescribing physician if and when it is determined that compliance has fallen off. Currently, as a result of drastic cuts in reimbursement and improvements in technology, HME/RT providers are scheduling fewer home service visits and tend to focus more on equipment performance than on patient compliance. It may well be time to seriously consider the possibility that LTOT will be terminated in those situations when continued noncompliance is detected.

In terms of medical justification, it may also be time to require a periodic reassessment of the clinical impact of LTOT and to link this reassessment to continued coverage and reimbursement. If, as has been suggested by Tiep and Celli, as many as 20–40% of hypoxemic COPD patients who initially qualify for LTOT may no longer meet coverage criteria after one month of therapy, the idea of periodically reassessing the need for LTOT warrants serious consideration.[26,27] However, if and when resting, ambient air, arterial oxygen tension returns to $>$ 60 mm Hg following a regimen of LTOT, close follow-up is essential if a decision is made to discontinue the oxygen.[28]

Respiratory therapists employed by HME/RT providers could easily assume an expanded role in this area and

provide the prescribing physician with specific and important information on a regular basis. However, under the current system, because of concerns over program integrity, HME/RT Medicare prohibits providers from providing laboratory evidence of eligibility for coverage.[12] Thus, any expanded role of HME/RT providers in measuring blood oxygen levels and using these results to determine continued eligibility for coverage would require revision of Medicare regulations. It is unfortunate that many highly qualified and reputable respiratory therapists and their employer providers are limited by archaic Medicare policy from developing innovative home monitoring and assessment programs that could greatly improve the link between the prescribing physician and the patient suffering from COPD-induced chronic hypoxemia.

Last, it is important that the continuing debate over the cost-effectiveness and economic contribution of LTOT not lead to the neglect of other proven treatment interventions for COPD.[29] Pharmacologic therapy, airway clearance therapy, and exercise conditioning have all been recognized as essential adjuncts to LTOT, and should not be relegated to a secondary position or ignored.[30,31] Unfortunately, LTOT tends to draw the majority of the attention, no doubt because of the size of the annual expenditure for this therapy. It is probably safe to assume that a successful program of LTOT is more the result of a combination of several proven therapies administered on a consistent basis than simply the effect of LTOT alone. Future decisions over who will pay for and who will be eligible for LTOT should be made in the context of what constitutes the most appropriate approach to manage COPD and the concomitant chronic hypoxemia.

Summary

Home oxygen therapy represents a scientifically validated and universally accepted therapeutic regimen for the treatment of chronic hypoxemia secondary to COPD. The clinical benefits of home oxygen, including a decrease in morbidity and often a concomitant increase in the quality of life have been repeatedly confirmed through rigorous worldwide trials, studies, and investigations. However, since home oxygen is an expensive treatment modality, important questions continue to be raised about the overall cost-benefit of the intervention. Such scrutiny is expected to continue, especially in the United States, as the entire issue of health care cost-containment remains atop the domestic political agenda.

Providers of home oxygen therapy have traditionally realized quite favorable reimbursement for home oxygen equipment, especially for those patient-customers covered under the Medicare program. However, recent Medicare reimbursement reductions of more than 30% have raised serious questions about the ability of home oxygen providers, especially those with annual revenues less than $1 million, to sustain their historical high level of support services to home oxygen patient-customers. Of particular concern is the economic hardship of supplying portable oxygen, especially for those patient-customers with unusually high ambulatory needs. The use of oxygen-conserving devices is viewed by some as one strategy to better control the costs of supplying portable oxygen, although there are those who still question whether or not oxygen-conserving devices can effectively forestall arterial oxygen desaturation across the entire spectrum of ambulation.

Given the evidence now being reported that compliance in using home oxygen as prescribed may well be much lower than originally believed, the time is probably right to revisit the role played by home oxygen providers in determining continuing need through the performance of periodic reassessments. Such reassessments, if designed according to prescribed and validated protocols and conducted by home respiratory therapists under orders of the prescribing physician, would be a valuable tool to ensure continued medical need and identifying noncompliance. This would help ensure that those needing and using home oxygen would continue to receive the benefit. At the same time, patient-customers who, for one reason or another, stop using their oxygen equipment despite repeated encouragement, would have the equipment removed. The net result would be that reimbursement dollars currently wasted on home oxygen equipment that is not being used could be reallocated for those patient-customers willing and able to use the equipment as prescribed.

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Discussion

Petty: One reason I suggested that there might be up to a million LTOT users is because of the addition of the VA, which I've been told may be 100,000 to 150,000. Do you have any information on that?

Dunne: Yes, the other 20% supposedly includes the railroad workers, the coal miners, and the VA. Under the VA, in most of the larger institutions that prescribe the home oxygen benefit, it only can be rendered through the pulmonary medicine department. So the VA a long time ago realized the importance of having qualified physicians govern who receives home oxygen.

Kacmarek: You mentioned that respiratory therapists in the home should be allowed to make some recommendations about continuation of therapy. Although I personally agree with that as a therapist, I am concerned about the recommendations. You just mentioned that many thousands of companies are providing home oxygen therapy in spite of the fact that the revenue is going down. Home oxygen therapy is still a highly profitable enterprise, and it seems to me there is an inherent conflict of interest if you're working for a company who can benefit from your saying that this patient still needs oxygen therapy. Although from the conceptual point of view I think it's the way to go, I don't see how it can reasonably be done in this environment where there are patients who are left on oxygen therapy who clearly don't need it. I agree, however, that there need to be regulations that mandate reassessment of whether patients meet the requirements for oxygen therapy, particularly that subpopulation who are placed on oxygen therapy after an acute exacerbation. My question is, of that 700,000 or more patients who are receiving oxygen therapy, are there data to differentiate the patients we've been discussing—stable COPD patients electively placed on oxygen therapy because they truly demonstrate the need for oxygen at a point of stability, compared to patients who are placed on oxygen therapy after an acute exacerbation. It would seem to me that, if that subpopulation placed on oxygen after an acute exacerbation were managed properly, there would be a lot of money saved to help deal with those patients who really need long-term oxygen.

Dunne: Several very interesting points. First, yes, my recommendation of the expanded role of the respiratory therapist is very controversial, but I know a lot of respiratory therapists in home care who have expressed to metheir frustration at not being able to do respiratory care in the home, but to basically only dance around it and to take care of equipment and do patient education, which is very important, but I think the respiratory care component is measuring, assessing, and looking at important functions—oximetry, blood gases, exercise tolerance, and perhaps even quality of life measures. We don't do that right now because the Medicare fee-for-service reimbursement process is not conducive for us to discontinue patients on home oxygen. But when you get into risk-sharing prospective payment, where basically you're pre-paid in advance of the service you render, your whole attitude changes. You try to make sure that you're not withholding it from those who need it and not giving it to people who don't need it. I think that's where the disconnect is.

Respiratory therapists have been involved in home oxygen when the margins were high because it was the right thing to do and made for better patient outcomes. And now I'm quite certain this industry is at a crossroads and that there will be a lot of casualties based on the cutbacks already in place. And, if there is another 20% reduction resulting from competitive bidding, there won't be that many providers left that are real serious players. I think those companies will then re-establish their role as being the definitive competitive provider in the marketplace, and the physician community will respond accordingly. In terms of the number of patients who come on service who are there for the short term because of their exacerbation, Medicare is attempting to handle this by requiring that the qualifying blood gas must now be done within 48 hours of discharge. At one time you would look for the worst blood gas, which was usually on admission. Unfortunately, the data from Silverman did not show what the diagnosis was in relation to the number of claims paid.[1] But they did look at the number of rehospitalizations, and COPD was the number one cause.

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O'Donohue: There are some data available on compliance in the United States that I think are very striking: specifically a 5-state survey done a few years ago by the Office of the Inspector General looked at patients who had been discharged from the hospital with oxygen concentrators.[1] They started in Georgia and then extended it to 4 other states because of their findings—that 50% of the patients were not using oxygen at the time of the survey. Almost none of the physicians had any formal records of the patients' being on oxygen therapy. As I recall, about a third of the physicians didn't know that the patient was receiving oxygen therapy, and an equal number did not know the regulations or the indications for home oxygen. Their conclusion was that the physician's role in home oxygen appeared to be "the signer of a Certificate of Medical Necessity."

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Dunne: Again, you underscore the point that often the prescribing physician is not involved with the sustained use of the equipment they are prescribing. Home care companies must therefore assume greater responsibility for compliance.

Wedzicha: Can I make an analogy to some of our experiences in the United Kingdom? Recently the Royal College of Physicians was charged by the Department of Health to look into prescription of oxygen therapy. What we came up with was a very important guideline, which is that oxygen must be prescribed by respiratory physicians or specialists. You also have the means to investigate and measure the blood gases. In the United Kingdom, currently, oxygen must be prescribed by primary care physicians. We may assess the patient, but the prescription has to be signed by primary care. So this is an area where there have been problems. We have recommended that all prescriptions must be specialist-led. The other point is about bidding. In the United Kingdom we have a system of bidding for system equipment. The oxygen concentrator costs have fallen. It is now about 50£ ($80) a month for an oxygen concentrator. The problem is that we had respiratory nurses/therapists going to patient's homes running a good service 7 or 8 years ago. Now we have no follow-up arrangements unless we organize them ourselves. Thus, the second recommendation in our report was that patients need some follow-up.

Greg Spratt:* In regard to the noncompliance problem, I don't think that rate is too dissimilar from what we see with every type of therapy that we provide, such as metered-dose inhalers or medications. I think it points to the fact that noncompliance is a problem in all forms of therapy, and education is a real need. I appreciate your comments in that they raise the issue of whether we are looking at this as simply a technology—a fast-food commodity almost—where patients, if we continue in our current path, will stop by and pick up a concentrator and be responsible for their own care. We should recognize the need for servicing these patients and try to come up with some workable method to see that they do receive the education and follow-up they need. I really think that we're at the crossroads in the United States right now. If we allow Medicare to have its way and simply look at it as a technology, the price will continue to drop, and the service will drop along with it. One solution that has been proposed is to separate the service component from the equipment component in terms of reimbursement and actually reimburse for the services of the follow-up, the education, and other service components. I believe that would be very cost-effective if we can improve the utilization of the therapy.

Dunne: With regard to the United Kingdom having only pulmonary specialists prescribe, when we alluded earlier to the present guidelines for noninvasive positive-pressure ventilatory assistance, the recommendation was that a pulmonologist had to drive that process. However, there was a loud hue and cry from nonpulmonologists, and I suspect the same would happen if it were suggested only a pulmonolo-

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gist could prescribe home oxygen—although it might well add serious clinical oversight that is presently lacking. What we are actually talking about is having a restricted panel of specialists who really know how to do it right. Greg, I think your point is well taken that there is a disconnect between the service component, the care component, and the technology—a direct result of flawed and antiquated Medicare policy. I suspect that we're going to have to see some fundamental changes in the Medicare process or programs as we know them. I think those are coming. I think if we were a "fly on the wall" in the hallowed halls where Medicare policy is being formulated today, we would see that they would do everything possible to get every Medicare patient to join a Medicare risk HMO the moment they become eligible. That way, Medicare is sending one check for each beneficiary once a month to one health plan: let the health plan worry about negotiating the subcontracting of the various services. Under current Medicare guidelines, I don't think we're going to see any change in getting reimbursement for the care component, whether it be for the respiratory therapist, the home health nurse, or anybody else for that matter. I think it is safe to say that we need to do something different. What has been done in the past is not working.

McCoy: On your comment about our being at the crossroads, we've talked about medicine here, but I think economics is driving our medicine on LTOT, and one of the things happening is that prices are coming down, but there are no standards of care. From a long-term oxygen standpoint, there are no standards, so Medicare or HCFA can keep going down, because there is no basement. They'll cut it, and the first response from the home care industry is that a 25–30% cut is going to limit access to oxygen. The General Accounting Office showed there wasn't a limit to access with a 30% cut. It's sort of gave them some more ammunition to keep moving forward, and they're talking about another cut. The General Accounting Office report looked at the VA model and said, "Why is the VA spending less than we are?" So they keep on looking at cutting, but there is no consequence. There is no basement. That's why I think from this group here, if we come up with standards that have an economic basis as well as a scientific medical basis, it will give ammunition that will direct and control the payers, at least in this country. This group needs to come up with the science, but also with what the consequences of not doing the science are in terms of cost of hospitalization.

Sam Giordano:* I'm glad you worked in the GAO report. It's important to note that its conclusions aren't consistent with the scientific literature. The issue, in terms of home respiratory therapy and oxygen therapy, is, first of all, there is a respiratory therapy Medicare benefit and respiratory therapists are not allowed to participate. Currently only physical therapists and registered nurses can provide respiratory therapy visits for Medicare beneficiaries. Even though we've been around for almost 50 years and are the only ones who are specifically educated in this area and tested for competency, for some reason, or for many reasons, we've not been included in the benefit. I think sending people into the home who, frankly, don't know what they're doing, feeds into misallocation. Jamie (Stoller) cited several studies in his writings about the misallocation of various respiratory therapy modalities, and oxygen therapy was on that list, as I remember. Now, the economic argument: certainly it's important to document the avoidance of more expensive health care events, such as emergency department visits and hospital admissions, but I think we have an opportunity—especially with the advent of clinical practice guidelines and protocols—to knock down misallocation and try to bring about appropriate balance between LTOT need and appropriate access to services and equipment to satisfy that need. That's not been done, and must be done by people who know what's going on in terms of both the technology and what the physicians want. Respiratory therapists are obviously well positioned as consultants in this role. Moving on to the separation of the provision of service with the assessment for the need of that service, we (the American Association for Respiratory Care) proposed 10 years ago that a study be undertaken to measure the impact of this approach. It was rejected in Washington because of a strong lobby from the dealer side of the equation. We can still demonstrate value. This is the opportunity that we must seize through collaborative outcomes research efforts. Finally, as far as compliance is concerned, I understand from the NAEPP (National Asthma Education and Prevention Program) that the compliance with NAEPP guidelines for the management of asthma among physicians hovers just below 50%. So I think this ties into what Greg (Spratt) said in terms of the noncompliance: even though it is not acceptable to us, it is not unusual in medicine.

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A Nationwide System of Long-Term Oxygen Therapy: The Polish Experience

Jan Zieliński MD

Introduction
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Eligibility Criteria
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Summary

[Respir Care 2000;45(2):231–235] *Key words: oxygen therapy, Poland, organization, tuberculosis, chronic obstructive pulmonary disease, interstitial pulmonary fibrosis.*

Introduction

The first written report on in-home oxygen use in Poland comes from the year 1926. A famous Polish poet and writer, Jan Kasprowicz, suffered from severe left heart failure and bouts of nocturnal pulmonary edema. The writer's wife, Maria, wrote in her diary: "During the fourth attack, oxygen was used for the first time. This gave Johnny some relief. I told him not to be afraid of balloons since similar were always used by Andzia's husband during his attacks of asthma."[1] Kasprowicz died in the summer 1926.

Up until the 1980s, oxygen in large cylinders was sporadically used at home to treat patients with severe hypoxemia. However, the cost of such treatment was high and not reimbursed by the National Health Service. Not many families could afford it. After Neff and Petty[2] published a paper demonstrating the benefits of continuous home oxygen administration in patients with cor pulmonale, the number of cor pulmonale patients treated with home oxygen increased.

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After two landmark controlled studies of domiciliary long-term oxygen treatment (LTOT) were published,[3,4] Polish pulmonologists increased their efforts to introduce LTOT in Poland. In 1985 a pilot study with the use of oxygen concentrators, the most economical oxygen source, was performed at the Institute of Tuberculosis and Lung Diseases in Warsaw. In the spring of 1986 I spent a week at The University of Colorado Health Sciences Center in Denver visiting Tom Petty and discussing with him practical points about LTOT. In the fall of 1986 the Ministry of Health in Poland approved LTOT as a treatment for severe respiratory failure. Between 1987 and 1991 the system was implemented nationwide (Fig. 1).

Organization

It was decided to incorporate LTOT organization into a net of tuberculosis (TB) and pulmonary disease clinics. Such a net was organized after World War II to control a TB epidemic that swept the country during and after the war. In every administrative part (province) of the country a TB clinic was opened. The work of all TB clinics was directly connected with the central institution, the National Tuberculosis Institute in Warsaw. The institute trained personnel from provincial TB clinics. New methods of TB diagnosis and treatment were adopted by the institute and quickly implemented all over the country.

Eventually, with TB control in the country, the system took care of all chest diseases. Now the main preoccupa-

Fig. 1. Map of Poland showing the locations of 49 provincial LTOT centers.

tion of pulmonologists is non-TB diseases of the respiratory system. The Institute of Tuberculosis and Lung Diseases conserved its leading role, introducing developments in respiratory medicine to respiratory physicians all over the country. Figure 2 shows the structure of respiratory care and LTOT in Poland.

The headquarters of LTOT at the institute was responsible for issuing guidelines of indications for LTOT and unified follow-up and care of LTOT patients. All physicians responsible for LTOT at the provincial level were trained at the institute's LTOT center and equipped with uniform inclusion and follow-up forms. Physicians working at provincial LTOT centers are certified pulmonary specialists.

Once a year, a two-day workshop is organized, gathering physicians and nurses from all the LTOT centers in the country. The workshop opens with lectures by invited speakers devoted to hot topics in respiratory care, followed by detailed reports from LTOT centers, with special emphasis on local difficulties. Every LTOT center regularly sends copies of eligibility forms of included patients to the Warsaw center and annually reports deaths of patients undergoing LTOT.

Eligibility Criteria

Qualified for LTOT are patients suffering from chronic respiratory disease leading to chronic respiratory failure. The main qualifying criterion is arterial partial pressure of oxygen (P_{aO_2}) $\leq$ 55 mm Hg. The stability of the patient's status should be confirmed by two measurements performed 4 weeks apart. If at the second measurement P_{aO_2} is better than the initial measurement by $\geq$ 5 mm Hg, a third measurement after another 4 weeks is required. The stability of the patient's status is additionally verified by spirometry performed at the same time as blood gas measurements. Improvement of the forced expiratory volume in the first second (FEV_1) and/or forced vital capacity at the second measurement also delays the qualification decision for another 4 weeks.

Also, patients with less severe hypoxemia (P_{aO_2} between 56 and 60 mm Hg) may qualify for LTOT if their hypoxemia is accompanied by two of the following: (1) signs of pulmonary hypertension on a chest radiograph, (2) signs of a right ventricle hypertrophy on electrocardiogram, or (3) elevated hematocrit (> 55%).

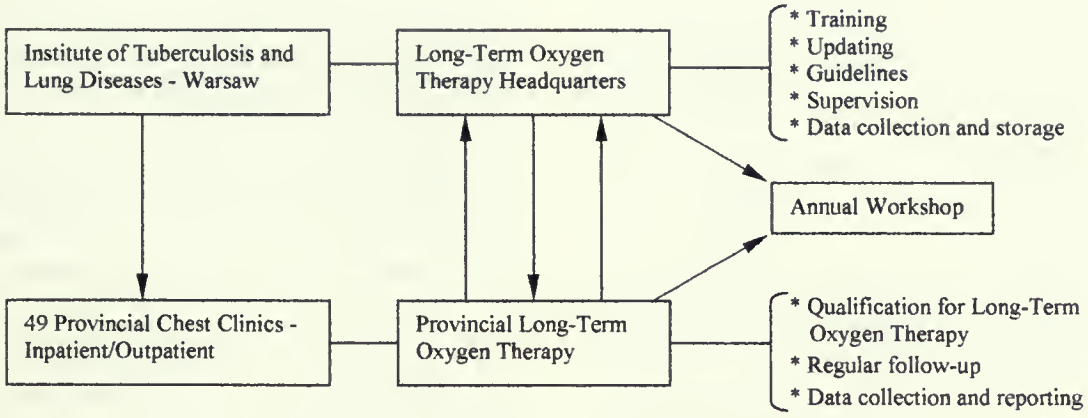

Fig. 2. Structure of the LTOT system in Poland. See text for explanations.

To be qualified, stability of hypoxemia has to be confirmed after a 3-month probationary period. At the introduction of LTOT in Poland, the upper level of P_{aO_2} qualifying for LTOT treatment was set at 65 mm Hg. After 5 years we decided to lower it to 60 mm Hg.

Patients with hypoxemia caused by heart disease or malignancy are not qualified for LTOT. Patients who continue to smoke are also not qualified. The nonsmoking status is verified by carbon monoxide measurements in the exhaled air and carboxyhemoglobin level in the blood, which should be below 3%.

We do not prescribe LTOT for smokers, for two reasons. First, because of elevated carboxyhemoglobin level in smokers, oxygen transport to the tissues is reduced.[5] In the situation of insufficient resources we would like to offer treatment to those who benefit the most. Second, smokers are at risk of burns.[6] Although the nonsmoking condition is respected at entry, about 10% of patients undergoing LTOT later resume smoking a few cigarettes daily.

Equipment

The LTOT system in Poland is based on oxygen concentrators. Several models manufactured in the United States have been used. Oxygen concentrators are purchased by provincial chest clinics and given to qualified patients free of charge. Maintenance costs are also covered by the National Health Service. Electricity costs are met by the patient. However, patients with a low income may have electricity bills reimbursed. Liquid oxygen is used only in big cities and on a limited scale.

Follow-Up

Patients undergoing LTOT are closely followed. They are seen at the center every 3 months. Medical examination is combined with blood gas and spirometric measurements. Every 12 months chest radiography, electrocardiography, and blood count and hematocrit are performed.

Nurse Visit

Once a month a patient is visited at home by a nurse from the center. She assesses the general status of the patient, checks daily oxygen use, and encourages the patient to use more oxygen if the oxygen breathing hours are less than 15 a day. Oxygenation is assessed via pulse oximetry while the patient breathes oxygen at the prescribed flow. The nurse also checks the concentration of oxygen produced by the oxygen concentrator. The older models of oxygen concentrator are not equipped with an oxygen concentration sensor and alarm. The nurse also changes filters at planned intervals and performs other minor maintenance procedures. This reduces the cost of maintenance from oxygen concentrator dealers. The nurse reports results of visits to the physician at the center.

We have found that the nurse plays a crucial role in the system. In addition to professional medical help, the nurse also helps patients with social problems, and sometimes is the only empathetic person contacting lonely patients.

Patients Treated

Originally around 70% of those receiving LTOT were chronic obstructive pulmonary disease (COPD) patients. The second largest LTOT population was patients with post-TB widespread pulmonary changes. Usually those patients also presented with signs of COPD. During the last few years the number of TB sequelae patients has diminished and the number of COPD patients increased to 80% of the total treated with LTOT in Poland. The second largest population now is interstitial pulmonary fibrosis patients. Table 1 shows the diagnoses of the first 1,000 patients who qualified for LTOT.[7]

Benefits of the System

The main benefit of the system is an accurate qualification procedure. This was especially important, consid-

Table 1. LTOT by Diagnosis, First 1,000 Patients Nationwide (1991)

COPD	67.6%
TB sequelae	9.0%
IPF	8.4%
Bronchiectasis	2.9%
Kyphoscoliosis	2.9%
Silicosis	0.4%
Others	8.8%

LTOT = long-term oxygen therapy; COPD = chronic obstructive pulmonary disease; TB = tuberculosis; IPF = interstitial pulmonary fibrosis.

ering the insufficient number of available oxygen concentrators. Accuracy of qualification was verified in some 400 patients in 12 provincial centers and found to be complying with guidelines in 100%.[8] We believe that this very good result is because qualification is assured by a specifically trained and instructed pulmonary specialist. Prescription of LTOT by general practitioners results in more than half of the patients being incorrectly qualified.[9,10]

National guidelines frequently are not observed by general practitioners. Waterhouse et al,[11] in a survey performed in England and Wales, found that only half of the patients undergoing LTOT had basal arterial oxygen saturation of $\leq 90\%$ at the start of treatment, together with a satisfactory improvement in arterial oxygen saturation on the prescribed flow of oxygen. Resources continue to be used for treating patients without ensuring that they benefit. The authors concluded that supervision of LTOT patients should be centrally funded.

Another important benefit seems to be an opportunity to study large, nonselected closely-followed groups of patients. We were able to perform original studies adding important new data to the daily practice of LTOT. One of the important questions we tried to answer was: should LTOT be withdrawn in a patient whose consecutive follow-up P_{aO_2} is above the qualification level? Górecka et al[8] followed a group of 400 patients for at least 3 years, looking for evolution of P_{aO_2} under treatment. They found that in patients qualified for LTOT ($P_{aO_2} < 55$ mm Hg), improvement of P_{aO_2} above 55 mm Hg was usually transitory. The further course of the disease in those patients was similar to patients in whom P_{aO_2} steadily deteriorated. In contrast, improvement of P_{aO_2} above 65 mm Hg in patients who qualified with moderate hypoxemia was usually permanent. LTOT in such patients may be safely discontinued, at least temporarily.

Other original investigations were prospective longitudinal observations of effects of LTOT on pulmonary hemodynamics.[12] Ninety-five COPD patients undergoing LTOT were followed for 6 years. Pulmonary hemodynamics were studied at 2-year intervals. This was the longest and largest study published so far on that topic. It was found that LTOT, after initial reduction of pulmonary hypertension, stabilized pulmonary arterial pressure over 6 years, despite deterioration in P_{aO_2} and increase in arterial partial pressure of carbon dioxide.

Perhaps the most important studies were prospective controlled investigations on effects of LTOT in patients with moderate hypoxemia. Górecka et al[13] studied 135 COPD patients who had P_{aO_2} of 56–65 mm Hg, advanced air flow limitation, and FEV_1 of 0.83 ± 0.28 L. They were randomly allocated to a control group ($n = 67$) or an LTOT group ($n = 68$). The patients were followed every 3 months for at least 3 years or until death. The cumulative survival rate was 88% at 1 year, 77% at 2 years, and 66% at 3 years. No significant differences were found in survival rates between the controls and the patients treated with LTOT, nor did longer oxygen use (> 15 h/d) improve survival. Younger age, better spirometric values, and higher body mass index predicted better survival. The authors concluded that domiciliary oxygen treatment does not prolong survival in COPD patients who have moderate hypoxemia. Airway limitation seems to determine survival in this group of patients.

A number of other studies performed in LTOT centers in Poland[14,15] or in cooperation with other centers in Europe[16–19] were published in international peer-reviewed journals.

The Future

The National Health System in Poland is undergoing an important reform. Beginning January 1, 1999, a centralized, budgeted type of health care has been changed for 16 independent regional Sick Funds. It was proposed that, instead of purchasing oxygen concentrators, the Sick Funds rent the equipment from specialized companies. Prescriptions for LTOT will be valid for 3 months. Qualification of patients and prolongation of prescriptions will be assured by provincial LTOT centers, as before. I am afraid that with the increasing labor costs in Poland, it will be difficult to continue nurse visits. On the other hand, the need for LTOT in the country will be satisfied in the coming 2–3 years.

Summary

Poland's Institute of Tuberculosis and Lung Diseases oversees 49 provincial clinics, which provide and monitor LTOT for patients with COPD, interstitial pulmonary fibrosis, and other pulmonary conditions. Because of limited resources, eligibility for LTOT is fairly strictly defined, and LTOT equipment is distributed to and retained only by nonsmoking patients who continue to demonstrate

need of the equipment (ie, those who have ongoing hypoxemia that can benefit from LTOT). This national LTOT system provides a large, nonselected population suitable for LTOT research, and recent studies have produced important data regarding survival, pulmonary hemodynamics, and the effect of withdrawing LTOT from patients whose oxygenation has recovered to above the LTOT qualification level of $P_{aO_2} \leq 55$ mm Hg.

ACKNOWLEDGMENTS

To Tom Petty, my mentor and friend. Also, my sincere thanks are due to Mrs Alicja Dymont for her invaluable secretarial assistance.

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Discussion

Pierson: I'll start it off by asking about the cost of electricity for the concentrator. My understanding is that it's about $50 a month for the electricity in the United States. Is that close? Also, my understanding is that it is provided directly by the government in France if you're on the national system. How is it in Poland?

Zieliński: In Poland, the electricity cost is much lower. It's some $20 per month, but of course you cannot compare this with the United States, because of the different economic status of the two countries. The electricity cost is normally on the patient side, but if the patient has a low income, he or she can claim to have this expense reimbursed.

***Sam Giordano:** Dr Zieliński, has any work been done to follow the hospital utilization of these patients?

Zieliński: Only one center did such a study, and they found that the hospitalization was two times lower after starting the oxygen treatment than it was before the LTOT. But no nationwide survey was done.

Dunne: During the monthly visits you described, is there any attention paid to the other forms of therapy that are part of the management of COPD, specifically, compliance with pharmacologic prescriptions, airway clearance techniques, or exercise conditioning?

Zieliński: This is mainly for use of drugs. It's checked. No formal rehabilitation is proposed, but we encourage all patients to move about as much

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as possible. When a nurse goes to the patient's home, she also encourages the patient to exercise, but not in a formal way.

Dunne: I guess my reason for asking is that I've noticed that several of the presenters at this conference have been impressed with the utility of FEV_1 as a predictor of survival. More importantly, I'm struck by the work of Howard et al, who argue that COPD patients ultimately suc-cumb to their airways disease.[1] I'm just wondering if we're maybe losing sight of the importance of airway clearance techniques and keeping the airways healthy when we start using long-term oxygen, which probably is not doing much to help the airways.

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Zieliński: I agree that for airways patency, clearance of the airways is important. However, I think that also the parenchymal part of the disease—that is, emphysema—has some effect on FEV_1.

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Long-Term Oxygen Therapy: Conference Summary

Neil R MacIntyre MD FAARC

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Summary

[Respir Care 2000;45(2):237–245] Key words: long-term oxygen therapy, mortality, morbidity, chronic obstructive pulmonary disease, ventilation, oxygen desaturation, ambulatory oxygen, air travel.

Introduction

This conference is the latest in a long line of American Association for Respiratory Care/American Respiratory Care Foundation conferences that have addressed many aspects of the respiratory care profession. I have been privileged to have participated in several of them and have found them always to be scientifically comprehensive, intellectually challenging, and a great deal of fun. I think it's fair to say that this conference has lived up to the high standards of its predecessors.

In past conferences, the summarizer has traditionally reviewed each of the presentations in the order they were given. I'm not going to deviate from that format. My approach will be to first review what the charge to that particular speaker was. Then, rather than simply summarize their talks, I will try to give you my "take" on the key points that were made and the key questions that were raised. Finally, I will conclude each review with a memorable moment that particularly caught my fancy. So, with that brief introduction, let us begin.

History

It's fitting that Tom Petty started this conference off with a history of long-term oxygen therapy (LTOT). Tom is one of the giants in this field and certainly one of the major reasons that oxygen therapy is so widely available today. His charge was to review the history of oxygen therapy from both scientific and economic perspectives. He started with the discovery of oxygen by Priestley and reminded us how it often takes people who "think outside the box" to make revolutionary discoveries (Priestley was a heretic who supported both the French and American

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revolutions). Tom then provided an entertaining chronology of the evolution of oxygen from a scientific curiosity into the effective therapy for hypoxemia we know today. He did this by noting the important contributions of Lavoisier, Barach, his own Denver group, and others that led to the landmark long-term oxygen clinical trials conducted by the United States National Institutes of Health Nocturnal Oxygen Therapy Trial (NOTT)[1] and the British Medical Research Council (MRC).[2]

The history of oxygen "dosing" was also reviewed. Indeed, it was Tom's group that provided much of the rationale for dosing oxygen according to arterial blood gas targets instead of merely providing some arbitrary flow.

Tom also looked at LTOT from an economic perspective. Estimating that as many as one million patients receive LTOT in the United States (a figure that was supported by several other speakers), Tom emphasized the high societal costs of this therapy, but pointed out that the benefits of LTOT can actually reduce health care needs (and thus costs) in many patients.

My most memorable moment of Tom's presentation was the introduction of a new outcome measure—"patient chart weight." His dramatic pictures showing how patient medical care needs decreased after LTOT (as indicated by the size of the medical charts pre- and post-LTOT) were something that even the most stubborn bureaucrat could understand!

Physiology

Dave Pierson followed Tom, and his charge was to review the causes of hypoxia and how hypoxia affects the body's metabolic functions. He started off with definitions of hypoxia (low tissue oxygen) and anoxia (absent tissue oxygen). Hypoxemia, however, was more difficult to pin down. Strictly speaking, hypoxemia should mean low oxygen content in the blood, but with this broad definition, anemia would also qualify as hypoxemia. As Dave pointed out, the clinical convention is to think of hypoxemia as low oxygen partial pressure in the blood, and the conference participants agreed that we would use this definition for our discussions. This struck me as yet another example of how imprecise we often are with our terms in medicine.

Dave proceeded to give a nice overview of what causes hypoxemia: decreased alveolar ventilation, decreased inspired oxygen, poor ventilation-perfusion relationships, and shunts. During the subsequent discussions, the concepts of diffusion limitation and short red blood cell transit times were also introduced as additional causes of hypoxemia in certain disease states.

The consequences of hypoxemia are cellular hypoxia and anoxia, which, if severe enough, lead to multiple metabolic abnormalities and the development of acidosis. The body compensates for hypoxemia in a number of ways, including increased red cell mass, increased cardiac output, and increased ventilation. Indeed, a question that has always fascinated me is why some patients with chronic obstructive pulmonary disease (COPD) have a blunted ventilatory response to hypoxemia (the bronchitic "blue bloater") while others have a brisk ventilatory response to hypoxemia (the emphysematous "pink puffer"). Could it be that obstructive disease location is important? Specifically, if disease is predominantly in the larger airways, with mucus production and cough, the brain seems to decide that reduction in ventilatory work is more important than maintenance of blood gases. In contrast, if disease is more distal in the lung, involving small airways and alveoli, the brain seems to decide that blood gases are more important than ventilatory work reduction.

The discussion then turned to the fact that, because ventilation-perfusion mismatching (as opposed to shunts) is the major mechanism for hypoxemia in COPD, supplemental oxygen would be expected to readily correct it. Indeed, in reviewing the NOTT study, 95% of the hypoxemic COPD study patients had their hypoxemia adequately treated with $\leq$ 3 L/min oxygen supplementation.[3] Dave then cited a clever analysis that combined the NOTT trial data with the data from nonhypoxemic COPD patients in the National Institutes of Health intermittent positive-pressure breathing trial.[4] This showed that the treated NOTT patients had survival curves almost identical to those in the intermittent positive-pressure breathing trial who were not hypoxemic. In other words, it suggested that if you get rid of this hypoxemia in COPD patients, patient mortality reverted to that of the patient who is not hypoxemic.

My most memorable moment of Dave's presentation was the picture of the "blue bloater" and the "pink puffer" side by side, dramatically illustrating the extremes of ventilatory response to hypoxemia I noted above.

Assessment

The conference then moved on to technical issues related to LTOT. Dean Hess started this discussion and was charged with reviewing the various ways to monitor and measure oxygenation. He began by defining direct arterial blood gas measurements as the "gold standard" for quantifying hypoxemia. It didn't come out until the subsequent discussion, but I think one of the key take-home points about blood gas measurements was the magnitude of the inherent variability of the technique. It was pointed out that various proficiency programs recognize that 10% variability is within the 95% confidence interval for test reproducibility. This means, for example, that a partial pressure of oxygen (P_{O_2}) measurement of 70 mm Hg could actually represent a P_{O_2} of anywhere between 63 and 77 mm Hg; a partial pressure of carbon dioxide (P_{CO_2}) measurement of 50 mm Hg could represent a P_{CO_2} of anywhere

between 45 mm Hg and 55 mm Hg. Dean also addressed the issue of using capillary blood gases as a substitute for arterial measurements. I agreed with Dean when he pointed out that there are a number of potential pre-analytical errors with this technique, and I doubt we're going to see it take over, at least in adults, in the United States.

"Point of care" systems are an increasingly popular way to monitor arterial blood gases, and these can either be indwelling sensors or sensors attached to the arterial catheter, which sample withdrawn blood and then return it to the patient. Obvious advantages are the rapid turnaround time and the fact that no blood need be lost from the patient. Correlation of point of care measurements with those of traditional analyzers is remarkably good, but the expense of these systems is a concern.

Dean spent a lot of time reviewing the performance of pulse oximetry, a measurement that many people see as the "fifth vital sign." He pointed out quite correctly that the correlation of arterial oxygen saturation measured via pulse oximetry (S_{pO_2}) and arterial oxygen saturation measured via blood gas analysis (S_{aO_2}) is heavily dependent on a number of factors and can often be quite poor. In reviewing Carlin's data,[5] for instance, a substantial number of both false positives and false negatives were observed in patients where $S_{pO_2} < 85\%$ was used to predict arterial partial pressure of oxygen (P_{aO_2}) < 55 mm Hg and $S_{pO_2} > 85\%$ was used to predict $P_{aO_2} > 55$ mm Hg. Dean rightfully reminded us that "bad data are worse than no data." I was also intrigued with a survey Dean presented[6] that showed how poorly health professionals understood the meaning of S_{pO_2}. Indeed, some even thought it was a measurement of ventilatory pattern! Quite a commentary on the "fifth vital sign."

My most memorable moment during Dean's presentation came during the discussion. I had asked the group if they had ever seen a routine "spot check" of S_{pO_2}, in the absence of any other clinical signs, detect something important before anything else would have detected it. I pointed out that back home I've got a standing offer of a steak dinner for the first person who can bring me a documented case of this and I extended this same offer to this group. Needless to say, my steak dinner offer remained unclaimed in Cancún, just as it has in Durham!

I wish we had had more time on this topic because we weren't able to address the issue of direct tissue monitors for hypoxia. Dean briefly mentioned the concept of gastric tonometry and I raised the issue of near infrared monitoring of the redox state of brain and other tissues. Perhaps this could be the subject of a future conference

Adverse Effects

Josh Benditt followed and was charged with reviewing the hazards of normobaric oxygen. He divided his comments into two categories: pulmonary oxygen toxicity and carbon dioxide retention.

I found it interesting that the first description of pulmonary oxygen toxicity was 100 years ago in an animal model exposed to a fraction of inspired oxygen (F_{IO_2}) of 0.8 for 4 days. Indeed, much of the data on the pathology and dose response of the lung to high oxygen concentrations has come from animal experiments. Extrapolation of these findings to humans is risky, however, as oxygen toxicity appears very species dependent. It's very easy, for instance, to make a rat oxygen toxic; it's much more difficult to make a baboon oxygen toxic. Josh picked an F_{IO_2} of 0.6 as a threshold for pulmonary oxygen toxicity in humans, and the group did not challenge that value. However, I still think that the toxic F_{IO_2} threshold (both concentration and exposure time) remains unknown. In this regard, I was fascinated by Josh's review of Tom Petty's study showing subtle changes suggestive of oxygen toxicity in autopsies of COPD patients exposed chronically to only low levels of supplemental oxygen.[7] On the other hand, I recall my own experience working with military jet pilots with thousands of hours of intermittent exposure to supplemental oxygen who showed no apparent abnormalities in routine pulmonary function testing.

Josh then addressed the fact that mild hypercarbia is common in COPD patients on LTOT. A relatively mild hypoventilation (perhaps due to a reduction in hypoxic ventilatory drive?) was originally thought to be the mechanism of this. Subsequent studies, however, have suggested that oxygen effects on regional perfusion in the lung and the Haldane effect in red blood cells may be more important mechanisms of mild hypercarbia in the stable COPD patient on LTOT.[8] Nevertheless, I think we all agreed that abrupt and excessive increases in supplemental oxygen for the decompensating COPD patient can shut off the respiratory drive and produce a respiratory arrest.

My favorite memory of Josh's presentation was the picture of the Sasquatch, a legendary creature of the Pacific Northwest. Citing Dave Pierson's skepticism of the importance of oxygen toxicity in the intensive care unit, Josh likened oxygen toxicity to the Sasquatch —"often discussed but rarely seen."

Equipment

Bob Kacmarek was the next speaker and was charged with reviewing the equipment used for LTOT. Bob reviewed and "scored" the 3 major LTOT systems: liquid oxygen, tank, and concentrator. He gave liquid oxygen high scores for F_{IO_2} capabilities and portability, but low scores for capital and delivery costs. He gave high scores to tanks for F_{IO_2} and flow capabilities, but low scores for delivery costs. He gave concentrators high scores for costs, but low scores for F_{IO_2}, flow rate, and portability. In the

end, Bob's scorecard gave the "win" to liquid oxygen, but by a close margin. Interestingly, during the discussion, issues of ease of use and safety were also raised as legitimate additions to the scorecard.

What I thought was really intriguing in Bob's presentation was the concept of using a concentrator to fill tanks or even liquid oxygen systems. If this could be done easily, it would go a long way toward making the portable liquid or tank systems as cheap as concentrators.

My most memorable moment with Bob's presentation was marveling at how, once again, he had taken a complex topic and reduced it to a simple "scorecard." As the summarizer, I was grateful for this!

Conservation, Costs, and Quality

Bob McCoy followed as the first of two "industry" speakers. I think it's important that we have industry representation, both unofficially and officially, at these conferences. They give a perspective to things that I think is vital for clinicians to appreciate.

Bob's charge, on the surface, sounded somewhat technical. "What is the role of oxygen conservation devices and how do they work?" But the underlying theme of Bob's presentation was probably much more important: addressing the whole issue of LTOT cost. Let's face it: the primary purpose of a conserving device is to reduce cost. Aside from the fact that a conserving device might reduce system weight, it has no physiologic benefit. I hadn't realized that it costs $35 to send a truck out and refill tanks. If you're doing that once a week, and you're only getting a little over $200 a month in reimbursement for it, I can see why the issue of oxygen-conserving devices becomes critical. This is an important point for clinicians to understand.

Bob went through the various methods of conserving oxygen. I thought the first one was particularly interesting: just set the prescription right! Lower flow (or discontinue it when you don't need it). Raise flow only when you need it.

Another approach to conservation is reservoirs. While effective, these devices have serious cosmetic problems. The demand systems appear to have considerable potential, but the issue of system sensitivity/responsiveness to patient effort is important. I was particularly intrigued with the idea of "charging" the demand system tubing with oxygen *before* the next breath, and was disappointed that no such systems exist. Bob emphasized that the transtracheal system is an excellent oxygen-conserving device. I hadn't known that there are 15,000 of them in use; I also hadn't known that unreimbursed replacement costs were such a barrier to wider usage.

Bob pointed out that there's a lot of variability in the delivery of these systems. For instance, a 2 L/min setting can range the inspired F_{IO_2} from 24% to 28%, depending on the device, the inspiratory time, the tidal volume, etc. That's a difference of about 28 mm Hg, if I did my mathematics correctly, and that's a sizeable variability. The message was that you've got to carefully pick the right conserving device for the right situation.

During the discussion, we started talking about the issue of qualified people going to the home to make sure that patients are using LTOT appropriately—that they're using it when they're supposed to be using it, and that they've got the right dosing regimen. Conceptually, these kinds of assessments should save money, for two reasons. First, the most cost-effective device/dose can be provided and adjusted as needs change. Second, improved patient compliance should result in less need for other health care services. Qualified respiratory therapists seem ideal for this role.

My memorable moment from Bob's presentation was a sobering one. While the presentation was technical, the underlying message involved the very basic conflict between cost containment and quality care that we face every day in this business of taking care of patients.

Effects on Mortality and Morbidity

Jerry Criner followed and was charged with reviewing the mortality and major morbidity benefits of LTOT. Similar to previous speakers, he referred to the NOTT and MRC trials to demonstrate the important mortality benefits of LTOT in patients with persistent hypoxemia. He also reviewed medical care costs (ie, hospitalization rates) in these trials and showed that there was a trend toward decreased hospitalization, but the trend didn't reach statistical significance. One question that always comes up in these kinds of analyses is that control group hospitalization rate may be lower simply because the sickest died off more rapidly in that group!

Jerry pointed out that in a Polish study (later reviewed by Jan Zieliński), patients with $P_{O_2} > 55$ mm Hg received no benefit from LTOT over 3 years.[9] So, the message here is that oxygen therapy works, but it only works in those in whom hypoxemia is an issue.

Jerry then reviewed a number of hemodynamic studies, looking primarily at the issue of pulmonary artery pressures secondary to hypoxemia. He showed that in the NOTT trial, continuous oxygen therapy reduced pulmonary vascular resistance more than nocturnal oxygen therapy. He also reviewed other data showing that abrupt withdrawal of oxygen in hypoxemic patients really wreaked havoc on the pulmonary artery circulation. He also showed data that the acute response of pulmonary artery pressure to oxygen wasn't much of a predictor as to who was going to benefit from LTOT.

The most memorable aspect of Jerry's contribution was the incredible depth and breadth of his presentation. Indeed, his performance prompted Tom Petty to remind us

of the definition of an academic pulmonary physician: "Someone who is long-winded about the short-winded"!

Neuropsychological and Quality of Life Issues

Our first European representative was Jadwiga Wedzicha. She was charged with reviewing the issue of neuropsychiatric impairment and LTOT. I found this to be an interesting discussion, as much for what I didn't learn as what I did learn. By that I mean that the neuropsychiatric field is a very difficult field in which to do rigorously controlled studies. The instruments that measure neuropsychiatric function are quite subjective, and there's an incredible amount of "noise amidst the signals." It is thus very difficult to do these kinds of assessments in sick patients, particularly over time, particularly when there are a lot of other things going on. I think it's clear that there is neuropsychiatric impairment in hypoxemic COPD patients, though it's difficult to separate whether this impairment is due to hypoxemia, chronic disease, high red blood cell effect, medications, the presence of a supplemental oxygen device that makes a patient "look" impaired, or the abnormal mechanics of the lung disease.

There are data from the NOTT trial that show improvement in neuropsychiatric function, and it seemed to be better in the continuous oxygen group than the nocturnal oxygen group, especially at the 12-month mark.[10] There are no other well controlled trials addressing this, however. Jadwiga was thus forced to review a number of studies using historical or case controls. LTOT in these studies didn't necessarily improve neuropsychiatric scores, although as she put it, oxygen may have stabilized these symptoms

My most memorable moment came during the discussion period. A number of conference participants argued that randomized trials were needed to see if LTOT had beneficial neuropsychiatric effects for hypoxemic patients with disease other than COPD (no data exist in this field). Jadwiga countered passionately that depriving such hypoxemic patients of supplemental oxygen just to prove how badly they would do neuropsychiatrically would be unethical.

Long-Term Oxygen Therapy in Disorders Other than Chronic Obstructive Pulmonary Disease

We closed out the first day with Jan Zieliński, whose charge was to look at the effects of hypoxemia and LTOT in patients without COPD. He made a number of interesting points. First, he emphasized that around the world there is quite a range of long-term oxygen utilization in different diseases. For instance, in the United States only 24% of patients on oxygen are non-COPD, whereas in Japan it's 61%, and in the Czech Republic it's only 6%. In Japan much of the LTOT use is driven by the management of post-tuberculosis patients.

Jan then did something that we don't often see at a conference like this: he presented new data! Not only new data, but a whole trial that has never been published. The trial involved 62 patients with restrictive lung disease followed over a 4-year period. These patients had total lung capacity measurements less than 80%, were hypoxemic, and were randomized to LTOT or control. The most impressive results were that there was a huge mortality (90% after 3 years) and that LTOT seemed to have no effect on it. Jan discussed some other nonrandomized data of interstitial pulmonary fibrosis and oxygen usage, and all showed substantial mortality in hypoxemic interstitial pulmonary fibrosis. With that kind of mortality, oxygen therapy is unlikely to have much of an effect. This brought us back to the discussion we had previously with Jadwiga; if you're going to justify oxygen use in these types of patients, you need to have something other than mortality data (eg, improved quality of life data or medical care costs) to justify the expenditure.

My most memorable moment was giving Jan the award for the best new study presented at an American Association for Respiratory Care/American Respiratory Care Foundation Journal Conference! With that, the first day ended.

Long-Term Oxygen Therapy or Noninvasive Ventilation?

The next day Jadwiga started off with a kind of different twist on things. Instead of addressing LTOT, she was charged with reviewing the strategy of using noninvasive positive pressure ventilation (NPPV), either as a replacement for or adjunct to LTOT. The concept underlying NPPV therapy in hypoxemic patients is that "resting" overloaded ventilatory muscles at night might improve blood gases and patient outcome. It is important to note that her charge was *not* to discuss obstructive sleep apnea or central sleep apnea, because these are conditions where noninvasive ventilation (or continuous positive airway pressure) clearly does have a role.

Jadwiga relied heavily on the study she co-authored with Meecham Jones that supported the use of nocturnal NPPV in patients with hypercarbic COPD, based on improved blood gas values.[11] She also reviewed some smaller studies from Leger[12] and Jones[13] that suggested that hospitalizations might also be reduced in this population with the use of a mask ventilator. But she also pointed out that there are conflicting data here. Lin's data, for instance, suggested that NPPV did little for this patient population long-term.[14]

This issue is particularly relevant in the United States, where a number of regulations on NPPV are currently

being formulated by various payers. For instance, the tentative Medicare reimbursement rules will provide for NPPV in COPD patients only if there is hypercarbia above P_{CO_2} 52 mm Hg. Moreover, NPPV in this situation is not to have a backup rate unless there's a sleep study verifying that backup rates are needed

My most memorable moment during this presentation was again supplied by Tom Petty. He challenged the basic tenet of nocturnal NPPV in hypercarbic patients—that getting the daytime P_{CO_2} lower was beneficial. Indeed, he pointed out that the body adapts quite well to "permissive hypercapnia" and that nocturnal unloading of ventilatory muscles might have little or no long-term benefit.

Nocturnal Oxygen Desaturation

Walter O'Donohue followed, with the charge of reviewing nocturnal oxygen desaturation (NOD—certainly a great acronym for a sleep syndrome!). His focus was on patients who do not have daytime hypoxemia but who develop hypoxemia at night. Walter discussed possible mechanisms: hypoventilation, smaller tidal volume, muscle hypotonia, decreased functional residual capacity, increased dead space, increased airway resistance, mucous production, and decreased cough, decreased ventilatory drive, and ventilation-perfusion ratio mismatching from atelectasis and the supine position.

The criteria to define NOD is controversial. Some examples include: "$S_{pO_2} < 90\%$ for $> 30\%$ of sleep time"; "nocturnal $S_{pO_2} < 85\%$ for > 5 min"; and "S_{pO_2} falls $> 5\%$ at night with daytime symptoms." Walter estimated from the data in the literature that, depending upon the NOD criteria used, as many as 25–45% of COPD patients may actually have NOD. If so, this would dwarf the current number of patients currently receiving LTOT—a daunting economic prospect! At least in some studies NOD is associated with elevation in pulmonary artery pressure. This probably is associated with mortality and may be related to neuropsychiatric dysfunction.[15]

Unfortunately, there are no good daytime predictors of NOD, so nocturnal oxygenation assessments are needed for the diagnosis. This raised the issue of routinely screening COPD patients for NOD. Walter, however, wisely pointed out that, since we don't really know the benefits of nocturnal oxygen therapy in these otherwise asymptomatic patients (see below), we ought to restrict our nocturnal assessments to those in whom we think it's likely to be clinically important: patients with right heart dysfunction, polycythemia, and other indications of significant chronic hypoxemia.

Walter pointed out that there are very few studies showing that giving oxygen to patients with NOD really changes things. There is some evidence that LTOT in NOD patients can reverse pulmonary artery hypertension.[16] This conceivably could translate into a survival benefit. There does not appear to be much change in neuropsychiatric function with LTOT in NOD patients, however. It thus appears that the role of oxygen at night in NOD patients is unclear. I came into this conference thinking it was pretty straightforward that if you saw somebody with NOD you ought to treat them; now I'm not so sure.

The memorable moment here was Walter "doing the mathematics." Specifically, if in fact 25–45% of patients with COPD really do have NOD and really should get nocturnal LTOT, the cost would be staggering. It was sobering to contemplate where this money should come from.

Exercise Desaturation

My talk followed, and my charge was to review the issue of exercise hypoxemia (exercise oxygen desaturation or XOD). I first reviewed the mechanisms for exercise impairment in chronic lung disease. Certainly exercise hypoxemia is one of them, but abnormal ventilatory patterns, dyspnea, and right heart dysfunction are all components of this as well. Oxygen clearly can improve exercise performance in hypoxemic patients by preventing hypoxemia. But oxygen also appears to benefit nonhypoxemic patients as well. The mechanisms of this are not entirely clear, but they appear to be related to decreased ventilatory needs ("permissive hypercapnia"), better ventilatory patterns (longer expiratory times reducing air trapping), and, in those with bad right heart function, improvement in ventricular performance. There may also be a reduction in subjective exercise-related dyspnea.

I next addressed the issue of whether these kinds of patients can be reconditioned if the XOD can be treated with oxygen. The study by Kramer addressed this.[17] He took hypoxemic patients to the "hyperbaric" conditions of the Dead Sea and showed that increasing the partial pressure of inspired oxygen improved exercise performance. Moreover, training these patients in this environment produced a conditioning effect that stayed intact even upon return to sea level. My own data on hypoxemic patients in a rehabilitation program support this notion.[18] In contrast, data from McDonald suggested that supplemental oxygen in these patients during activities of daily living (not exercise training) had little long-term benefit.[19]

My most memorable moment during my presentation was that I didn't have to take excessive notes for this summary!

Ambulatory Oxygen

I thought it was appropriate that we had Tom Petty come back after me to discuss the issue of ambulatory oxygen. I say this because ambulatory oxygen is required if we expect hypoxemic patients to safely perform meta-

bolically-challenging activities. Tom brought the NOTT trial back, but he analyzed it in a different way this time. He showed that those on continuous oxygen therapy who *walked*, who walked a lot, had by far the best outcome. This analysis, of course, raises the question of what came first (ie, was it those who *could* walk had better outcomes or was it that exercise made possible by oxygen was the difference?). Nevertheless, I believe that if oxygen enhances the ability of a patient to increase activity, this has to be viewed as beneficial.

This whole issue is particularly critical because, as Bob McCoy and Pat Dunne addressed in their presentations, reimbursement for ambulatory oxygen is woefully underfunded. From the United States Health Care Financing Administration's perspective, it is much cheaper to just stick a concentrator alone in a home than it is to supply systems that allow portability and ambulation. This strategy provides a disincentive to the very thing we are trying to achieve—maximizing functional capabilities. Tom certainly provided the ammunition to get this re-thought!

Another interesting point that Tom brought up was the idea of "restorative effects" of oxygen. This means that after a number of weeks or months of oxygen usage the need for oxygen may actually go down. Whether this is because of better heart function, better conditioning, or some other mechanism is not clear. Regardless of the mechanism, Tom made the point that, just as you don't withdraw anti-hypertensive medicines from a person whose hypertension is under control, so also you should not remove oxygen from previously hypoxemic patients

I had two memorable moments during this presentation. First, Tom showed us a publication called *Oxygen Magazine*, which had attractive models cavorting on the cover. It was unclear how oxygen produced the results we were shown, but all of us clearly wanted subscription information! Second, Tom coined a new word, "schleppable," describing the difficulty an emphysema patient has in hauling oxygen tanks around.

Air Travel and Oxygen

Jamie Stoller followed with a discussion of the effects of altitude on hypoxemic patients, particularly with relationship to the safety of air travel and the logistics of air travel. This was a typical Jamie presentation—very comprehensive, very clear, and quite entertaining. He pointed out that commercial airlines are generally pressurized to an altitude equivalent of 6,000–8,000 feet. Knowing this, you can predict a patient's P_{O_2} at altitude with various formulas, but he emphasized that: (a) it is often difficult to predict a person's ventilatory response (and thus P_{aO_2} response) to altitude, and (b) the human body has remarkable tolerance for brief periods of moderate hypoxemia.

Jamie then described a number of techniques to simulate altitude in patients, in an effort to determine who might really need supplemental oxygen while flying. These included various mask setups as well as hypobaric oxygen exposure in a chamber. He pointed out that the Aerospace Medical Association recommends that patients ought to get oxygen while flying if the predicted or measured P_{O_2} at altitude is going to be less than 50 mm Hg.[20] I suspect, however, that a lot of people don't follow that recommendation (it would mean, for example, that most patients with resting P_{O_2} values in the low 70s should get supplemental oxygen while flying).

Interestingly, Jamie pointed out that there are still only about 1 in 30,000 flights with medical "incidences" and only about 10% of those are respiratory-related. Jamie also pointed out that there are about 70 to 200 oxygen patients on each airline each year. In reviewing airline oxygen policies, Jamie noted that 24% of airlines simply don't allow it. In those that do, cost can be prohibitive. Although some airlines will let patients have oxygen for free, most charge a fee, some as much as $1,500, with a requirement for up to 4 weeks of preparation.

Jamie brought forth two other issues about air travel that I found intriguing. One was that a benefit of going to altitude was that the slightly lower density breathing gas may help the ventilatory process. Another issue was that of blebs, or recent lung surgeries, where the hypobaric conditions of flight can cause expansion of closed gas spaces, with resulting pain or even lung rupture.

During Jamie's presentation my most memorable moment occurred during his review of airline policies on supplemental oxygen. In this review he pointed out that most airlines, out of concern for aircraft safety with an oxygen system, mandate that patients use the airline's equipment. Indeed, Jamie could find only one airline (ironically, the one airline that flew many of us down here) that allowed a patient to *bring his/her own tanks* on board. It was not surprising then to see several conference participants on the telephone rearranging flights following his presentation!

Demographics

Patrick Dunne, our second industry speaker, was charged with reviewing how many people are on LTOT, what are the "drivers" for providing LTOT, and what are the economic consequences of LTOT. He first "went through the mathematics" of how many people were on LTOT and finally arrived at the conclusion that the number in the United States was in the neighborhood of three quarters of a million, maybe as many as a million (remarkably close to Tom Petty's estimate of one million at the beginning of the conference!). A more interesting number, however, was that in the United States there are 241 LTOT patients

per 100,000 population, whereas in the rest of the world there are fewer than 30 per 100,000. That's an 8-fold difference in oxygen use, and probably reflects a combination of higher awareness, higher health care accessibility, and higher reimbursement in the United States.

Patrick then went through a nice review of the costs of LTOT. For instance, I didn't realize it costs $35 a month in electricity to run a concentrator or that the trucking costs for tanks/liquid oxygen are so high. Despite these, I thought it was interesting that Patrick still considers oxygen a high margin item (although portable systems, in particular, were threatening this margin). Pat pointed out that 4 months of LTOT costs about as much as 1 day of hospitalization (recall Tom Petty's "chart weight" outcome measure noted above). Pat, however, also pointed out a basic problem inherent in our governmental involvement in health care: Part A Medicare has little to do with Part B Medicare in terms of accounting or accountability. There is thus no incentive for development of programs in one that produce cost savings in the other

Following Pat's comments, there was a lively discussion about oxygen as a commodity versus oxygen as a therapeutic strategy. Issues regarding the need for technical expertise in setting up oxygen and proper assessment thereafter were again raised, and the potential role of the respiratory therapist was again emphasized. Bob McCoy made the point that without accepted standards there's "no basement" to the payment reductions that can take place.

My memorable moment during Pat's talk was listening to him describe an oxymoron as Health Care Financing Administration's ability to exercise "inherent reasonableness"!

The Polish System

The conference's last speaker was Jan. His charge was to summarize how his country (Poland) had addressed the issue of LTOT. Interestingly, Poland has LTOT-qualifying criteria similar to those of the United States, yet utilization is severalfold less than the United States. This again raised the issues of awareness, access to care, and availability of suppliers as major factors in utilization.

The Polish LTOT system provides only concentrators (no ambulatory systems), but Poland provides these patients with regular assessments, which include arterial blood gas analysis, spirometry, chest radiographs, and even nursing visits to the home. The argument could be made that these careful assessments might be an important factor in controlling utilization by assuring appropriateness. Jan also supplied data from the Polish system that suggested a significant drop in hospitalization rates for hypoxemic patients on LTOT.

My memorable moment during Jan's talk was his description of the home visit by the nurse. The Polish custom, apparently, is to not only have the nurse perform clinical assessments but to also to share a cup of tea with the family. A lot can be said about a system that values that kind of patient-caregiver involvement.

Summary

With Jan's presentation, the conference concluded. In looking back on it, I think it is obvious that the group heard a very comprehensive, state-of-the-art review of this very important topic. Obviously, LTOT has enormous clinical and financial impact for millions of patients around the world. Indeed, LTOT is one of the few therapies available that has clearly been shown in randomized controlled trials to impact mortality. There are many questions that remain, however. To me, the most important of these questions involve the diagnosis and management of patients who do not have resting hypoxemia but who do have NOD and/or XOD. How aggressively should we "screen" for these conditions? If we find them, do we treat continuously or just during the hypoxemic episodes? What is the role of supplemental oxygen during rehabilitative exercises (including usage in patients who don't become hypoxemic)? The answers to these questions will clearly have substantial clinical and financial impact.

Other memorable aspects of this conference included the tireless efforts of Ray Masferrer to pull this conference off, the special camaraderie of the participants that made the discussions so productive, and the lovely location that gave the conference an atmosphere of high quality. I'd like to recognize and thank two important groups. First, the American Association for Respiratory Care did a superb job of organizing the conference and providing the journal RESPIRATORY CARE as a forum to publish the proceedings. Second, our 3 industry sponsors not only provided critical funding support but also gave the group important perspectives during many of the discussions. These kinds of industry-profession collaborations benefit everyone. Finally, I'd like to extend my congratulations to all the speakers for jobs well done and to thank them for making my job as summarizer an enjoyable one.

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Listing and Reviews of Books and Other Media. Note to publishers: Send review copies of books, films, tapes, and software to RESPIRATORY CARE, 600 Ninth Avenue, Suite 702, Seattle WA 98104.

Books, Films, Tapes, & Software

Respiratory Care Patient Driven Protocols. Jan Phillips-Clar RRT, Richard M Ford RRT, Timothy Morris MD, David M Burns MD, University of California San Diego, Respiratory Services. Dallas, Texas: Daedalus Enterprises. 1998. Soft-cover, 198 pages, $85 for American Association for Respiratory Care members, $95 for nonmembers.

Respiratory Care Patient Driven Protocols provides a collection of policies, procedures, and algorithms for 9 respiratory therapy modalities (oxygen therapy, pulse oximetry, incentive spirometry, chest physiotherapy, autogenic drainage, aerosol therapy, intermittent positive-pressure breathing, positive end-expiratory pressure, and extubation), several variations of these modalities, and 4 brand-name-specific devices for therapy (Percussionaire, Flutter Valve, ThAIRapy Vest, BiPAP). Included in the above are 8 protocols modified specifically for use in the intensive care unit. These protocols are intended as models for respiratory therapy departments that are in the beginning stages of implementing respiratory care protocols. The book is written for respiratory therapists, physicians, and nurses who are or are planning to be involved in caring for patients receiving protocol-directed respiratory care.

The material is written in a combined narrative and outline form. The introduction emphasizes the need for respiratory protocols as a mechanism for improving the appropriate use of respiratory care services, and stresses the importance of physician-therapist interaction when developing and utilizing the protocols. Each protocol begins with the policy for the protocol (similar in content to the American Association for Respiratory Care [AARC] Clinical Practice Guidelines), followed by the associated algorithm and a procedure. The authors remind the reader to use the protocols presented in the book as guidelines for protocols that must be modified to fit the needs of individual facilities. The policies are complete in regard to indications for therapy, contraindications, hazards, complications, outcomes, justification for discontinuing, and "boundaries/interactions." Boundaries/interactions spell out specific criteria that indicate the need to contact a physician and notify the nurse.

Respiratory Care Patient Driven Protocols provides detailed examples of a method for writing respiratory care protocols that are suitable for a policy and procedure manual. The protocols modified specifically for use in the intensive care unit are a useful addition to the numerous existing protocols for regular nursing floors. Examples of the patient assessment/evaluation format and the patient care plan would be valuable contributions to the book. In addition, the reference list would be more complete if references regarding the rationale for implementing respiratory care protocols were included.

Although the material is understandable to experienced respiratory therapists, there are some abbreviations used that are not explained (eg, "RCP" in the introduction, and "PTE" in several protocols). There are a number of typographical and grammatical errors (eg, "the patient will be discontinued on oxygen," "if the patient is not capable to administer a MDI," "the therapist will communicate the patient care plan with the MD/nurse," and "other forms other than specified"). One notable error in the extubation algorithm is "change patient to *HiFi* neb if requiring $F_{IO_2} > 0.60$." I mention this with some reservation, realizing from experience how difficult it is to render algorithms error free.

This soft-cover protocol collection carries an $85 price tag for AARC members and $95 for nonmembers. Although a large amount of the information necessary for developing respiratory care protocols is available in the literature and through the AARC it may be well worth the price for respiratory care departments in the beginning stages of implementing respiratory protocols to have these examples collected and formatted for use as guidelines. Facilities with established respiratory protocols might also find this collection useful for modifying existing protocols or adding new protocols.

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Principles Of Pulmonary Medicine, 3rd edition. Steven E Weinberger MD. Philadelphia: WB Saunders. 1998. Soft-cover, illustrated, 385 pages, $39.

The third edition of **Principles Of Pulmonary Medicine** is an excellent introduction to pulmonary pathophysiology and clinical pulmonary medicine. The ideal reader of this book is the first-time student of pulmonary pathophysiology and medicine. However, the book is structured in a manner that makes it an appropriate review source for the nonphysician pulmonary care professional.

I read the first edition of this book in 1989, as a second-year medical student, during my pulmonary pathophysiology course. I was delighted then by the ease with which I could understand the book's content. In reviewing the third edition as a pulmonologist, I am still struck by the wonderful manner with which the book goes about explaining pulmonary pathophysiology and the basics of clinical pulmonary medicine.

The author uses several techniques to aid the reader. Marginal notations are used throughout the book to highlight important points. This format also allows for ease in browsing and reviewing. An ample number of figures are present to clarify points, and pictures of radiographs provide clinical correlations. Last, three appendixes are present. They contain sample questions and answers in the subject areas of pulmonary physiology, pulmonary function testing, and arterial blood gas analysis.

The first portion of the book is devoted to pulmonary physiology. Critical concepts are presented in a concise and easy-to-understand manner. The level of detail is appropriate for all except the diehard physiologist. Another chapter is devoted to the evaluation of the patient with pulmonary disease. Specifically, discussion regarding common physical exam findings is presented. In addition, the basics of chest radiograph interpretation are reviewed, along with brief explanations of commonly used imaging procedures (computed tomography, magnetic resonance imaging, ventilation-perfusion ratio scan, and pulmonary angiography). Last, this packed chapter addresses in a cursory manner the areas of pulmonary

function testing, arterial blood gas analysis, and pulse oximetry.

The majority of the book is devoted to clinical pulmonary medicine. Chapters present the etiology, pathology, pathophysiology, clinical features, diagnosis, and treatment of various cardinal pulmonary diseases. The depth of discussion provides a good overview of each disease process. Chapters address obstructive and interstitial lung diseases, diseases of the pulmonary vasculature, pulmonary malignancies, pleural diseases, and infectious diseases of the lung. Updating of various therapies (with references) has occurred in this edition. However, I was somewhat disappointed in several of the treatment sections. Specifically, the level of discussion was often too vague and, occasionally, outdated.

In summary, it was not by accident that I read this book from cover to cover as a medical student. Revisions and updates have only added to the value of this book. I recommend it without reservation to those seeking a similar level of understanding of pulmonary medicine.

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Gerontology for the Health Care Professional. Walter C Chop MS RRT and Regula H Robnett MEd MS OTR/L. Philadelphia: FA Davis. 1999. Soft-cover, 355 pages, $21.95.

Gerontology for the Health Care Professional contains chapters written by various health care providers, woven into a highly readable text that serves as a resource to providers and consumers. It is a good review for physicians and nurses, and a basic text for other disciplines.

Each chapter begins with a helpful outline and list of objectives. I found the list of keywords to be redundant, but the self-study quizzes, lists of learning activities that generate further discussion, and extensive references are helpful. However, many citations seem outdated and are prior to 1994.

The first two chapters explain demographic trends and the social aspects of aging. Early in the twentieth century only 4% (3.1 million people) of the total United States population was over 65 years of age. The phenomenon of the "graying of America" began accelerating as Americans born in 1946 turned 50 years of age. Since 1996, one American has turned 50 every 7 seconds. Projections to 2030 predict an ever-increasing elderly percentage of the total United States population, with an expansion to 22% (70.2 million people) over 65 years of age. Before 1900, people were often considered "old" between 40 and 50. Many feel that health status determination should be done by biological age, rather than chronological age, as everyone ages differently.

Ethnic aging around the world is extensively examined in Chapter 2. Dr Robert Butler's term "ageism" is defined "as a systematic stereotyping of, and discrimination against, people who are old." False myths of aging, finances, lifestyles, and changing family roles are explored.

Chapter 3, "The Physiology of Aging," and Chapter 4, "The Psychological, Behavioral, and Cognitive Aspects of Aging," provide concise explanations of various theories of aging, age-related changes in body systems, personality development, types of intelligence, dementia, and depression. Suicide, death, and bereavement issues are also discussed.

Chapter 5, on nutrition, emphasizes early screening and intervention for elderly at risk. There is a basic nutrition overview. Chapter 5, on drug therapy, is an excellent resource. It discusses pharmacokinetic changes, polypharmacy, noncompliance issues, and medication management. Chapter 7, on sexuality and aging, covers often overlooked topics well presented here. There are tables of presenting problems regarding sexual dysfunction, including drug-induced causes and potential solutions.

Chapter 8, "The Continuum of Care," explains choices from home health to nursing home placement. Chapter 9, on financing health care, makes sense out of a very complicated system of Medicare, Medicaid, and other private and government insurance plans. Chapter 10, on health care providers, defines the various roles and would be very helpful to consumers. Chapter 11, "Future Concerns in an Aging Society," completes the text by offering discussions of worldwide aging, financial and social issues, demographic shifts, and generational equity (younger people financing older people).

With the aging of our society, everyone should have information available on elderly wellness, illness, and social/financial issues related to this group. I highly recommend this text to consumers and providers.

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Evidence-Based Health Care Workbook and CD-ROM 1999. Critical Appraisal Skills Program. Oxford UK: Update Software Limited. 1999. Soft-cover, 76 pages, UK £34.95; US $56.

Systems requirements: Microsoft Windows PC with Windows 95 or NT, 16 MB RAM, hard disk with 54 KB free space, CD-ROM drive, Internet connection with Internet Explorer. Orders and inquiries: Update Software Ltd, Summertown Pavilion, Middle Way, Oxford UK OX2 7LG. http://www.update-software.com.

Over recent years health care expenses have come under closer scrutiny by agencies and companies that pay the bills. As a result, those who make decisions or seek to influence decisions about health care are required to assume greater responsibility for ensuring cost-benefits of the care; that is, the benefits must justify the cost of the care. The discovery of the benefits ascribed to health care interventions requires evidence, which is generated only through valid scientific research. This discovery process has given rise to the term "evidence-based health care."

Given the importance of basing health care decisions on evidence, there is a need for individuals in health care to learn valid methods in finding and appraising scientific evidence. The course materials reviewed in this workbook and CD-ROM aim to teach the fundamentals of this process, with a focus on methods for systematic reviews of medical literature. The learning program, called the Critical Appraisal Skills Programme, is best described as a tutorial, and is divided into 5 modules: (1) Introduction, (2) Asking the Question, (3) Finding the Evidence, (4) Appraising a Randomized Controlled Trial, and (5) Appraising a Review.

The introduction describes the course materials and how to use them. This includes how to navigate around the CD-ROM, which is quite simple and straightforward. Subsequent sections guide learners through the processes of generating and then answering questions about health care evidence.

Question generation is the subject of the initial substantive section. This section takes the learner through the steps in generating questions about the effectiveness of medical interventions, breaking the question into 4 operational components: (1) the patient or problem, (2) the intervention, (3) the control, and (4) the outcome. This section, like all the others, is fortified with apt examples and practice applications.

The section on "Finding the Evidence" describes the major databases for medical literature, such as MEDLINE, EMBASE, and the Cochrane Database of Systematic Reviews. The tutorial instructs on literature search techniques such as selecting search terms, and describes the importance of sensitivity in searching, as well as techniques for increasing sensitivity and specificity. A practice exercise guides the learner through a MEDLINE search.

Appraisal of trial quality is an important component of systematic literature reviews. Indeed, decisions on health care ought to find basis in *high quality* evidence rather than just *any* evidence. Adopting the randomized controlled trial as the "gold standard," the section on appraising randomized, controlled trials describes the essential elements of a randomized controlled trial, discusses each element, and explains how to evaluate it. The tutorial guides the learner through a set of questions to ask about each trial under consideration. The learner uses an example of an actual trial (which is in the workbook), for the appraisal exercise. In addition to randomization, the tutorial addresses issues such as blinding, selection bias, and control for dropouts. Finally, this section addresses the issues of clinical importance of results and their local applicability.

The next section is about appraisal of literature reviews. Health care personnel may tend to accept the validity of the results of a review without recognizing whether or not a review is biased. Therefore, this is an extremely important section, which also teaches systematic reviewing methods. Like the trial appraisal section, this one also uses an article in the workbook as a reference tool as the learner proceeds through the section. Issues addressed include whether the question is clearly focused, whether there was bias in the literature search, and whether the review assessed trial quality.

The review appraisal section clearly illustrates and explains the "blobbogram," which is a kind of graphic display common to systematic reviews of the meta-analytic variety. Statistical issues are also discussed here, particularly from the standpoint of appraising meta-analytic methods and whether results from trials are aggregable. Finally, the tutorial addresses cost-benefit analyses associated with systematic reviews, that is, the "bottom line" for a review.

Statistics is a subject that tends to induce fear in many professionals. However, the statistics section in this tutorial teaches about relevant statistics in a clear and simple manner. The section explains statistics such as odd ratios, risk ratios, and number needed to treat. It also explores and explains issues related to statistical significance of results.

Additional features of the CD-ROM include Internet links, a comprehensive glossary, and practice exercises. The exercises in the CD-ROM tutorial refer to randomized, controlled trials and systematic reviews in the workbook as examples. Learners use these examples to answer questions in the tutorial that aim to teach trial and review appraisal techniques.

Although some of the ideas addressed by Critical Appraisal Skills Programme are complex, the presentation is in language that health care practitioners and students should understand. The terms are explained in plain language, and even the statistics are clearly presented. The tutorial is moderately interactive and the screens are attractive. The overall presentation is highly professional.

I highly recommend the package for anyone interested in evidence-based medicine. It is suitable for nearly all levels of health care professional, as well as moderately educated laypersons. The program would be a useful adjunct to basic research courses in all health care curricula. I believe students will enjoy and benefit from the tutorial. My estimate is that a thorough progression through the entire program, including the practice exercises, would take a novitiate learner between 3 and 6 hours. However, I would recommend that the learner undertake one or two sections at a time.

One minor criticism of the tutorial is that it is not possible to skip over material. Some of the screens appear rather slowly and, if one wishes to skip over sequences, this can be a bit frustrating. A word of caution regarding the CD-ROM is that it loads very sluggishly unless the computer and CD drive are very fast. I loaded it on a 166 MHz PC with a 10X CD drive and rebooted twice because I thought that my computer had locked up. It had not locked up; I simply did not exercise the proper patience.

The package, which includes the workbook and PC-compatible CD-ROM, costs £34.95 (about $56) plus shipping, for 1–9 units. The price for each decreases as quantities increase. Currently, there is no provision for a network agreement.

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How to Report Statistics in Medicine: Annotated Guidelines for Authors, Editors, and Reviewers. Thomas A Lang MA and Michelle Secic MS, with a foreword by Edward J Huth MD. Philadelphia: American College of Physicians. 1997. Softcover, illustrated, 367 pages, $39.95.

This book consists of annotated guidelines to assist authors, editors, and reviewers in assuring that the correct and accurate information is included in published research papers. This book is meant to be a reference and is not meant to be read from cover to cover, although that is how I approached it for this review. The intended audience is stated in the title as being authors, editors, and reviewers. However, anyone interested in critically evaluating the research literature would find this book helpful. The authors' stated purpose in writing this book is "to provide a set of detailed, comprehensive, and understandable guidelines for reporting statistical information in medicine." This is not a statistics textbook. The authors do not discuss how to choose the appropriate test or how the statistical tests are calculated. These areas are left out on purpose, and the authors state that their goal was to develop guidelines that address only the "presentation of statistical information in scientific publications and [to] discuss some related concepts." These goals are reached with easy to read and easy to understand chapters and guidelines.

This book is well organized and it is easy to find the information you need. I did not find any errors in content, nor typographical errors. The guidelines presented were all easy to understand, straightforward, and well written. The more difficult guidelines are illustrated with clear examples. The authors also included additional statements that augment the guidelines. There are subguidelines,

potential problems, methods of checking, and related information statements, each one keyed with a different symbol. The authors devote several pages at the beginning of the book to discussing the difference between statistical significance and clinical importance. They refer to this important distinction several other times in several of the chapters.

Part 1 consists of 15 chapters. Each chapter addresses a different aspect of biostatistics and develops guidelines of what should be in each section of a research paper if this particular biostatistics concept were used in the paper under consideration. For example, Chapter 4 discusses the reporting of p values. The chapter outlines, in the form of guidelines, what should and should not be included about p values in a paper's introduction, methods, results, and discussion sections.

Part 2 of this book is a "Guide to Statistical Terms and Tests." It consists of mainly a glossary of terms used in the book, including the statistical tests mentioned in Part 1. The definitions of the statistical tests include brief descriptions of the types of variables involved, the purpose of the test, and a brief outline of how the results from the test should be reported. I would have found this section easier to use if the authors had included the statistical tests in a separate section and maybe expanded on that section to discuss the conditions for selecting each test. That would have added slightly to the size of the book, but it would be useful to the reader.

Part 3 repeats all the guidelines again in an "unannotated" referenced list of the guidelines. The references are to the literature where these guidelines are discussed. For example, Guideline 1.14: "Specify how the sample size was determined. If the sample size was determined in combination with a power calculation, give the details of the calculation." This guideline is followed by a list of 25 references in the "Author, Year" format, using only the first author's last name. This section is a compact list of all the guidelines, without any subguidelines, potential problems, methods of checking, related information statements, or examples.

Part 4 is the Appendixes section. Included here are sections on checklists for reporting clinical trials, mathematical symbols and notations, rules for presenting numbers in text, and the correct spelling for statistical terms and tests. The bibliography section is concise, appropriate, and recent.

In conclusion, this is a well written, easy to understand, and concise discussion of guidelines that should be followed in writing research papers for the medical literature. This book should be on the shelf of all authors, editors, manuscript reviewers, and anyone else interested in critical evaluation of the medical literature. If the authors writing for the medical literature would follow these guidelines, the editors and reviewers of many medical publications would have an easier time in evaluating manuscripts for publication.

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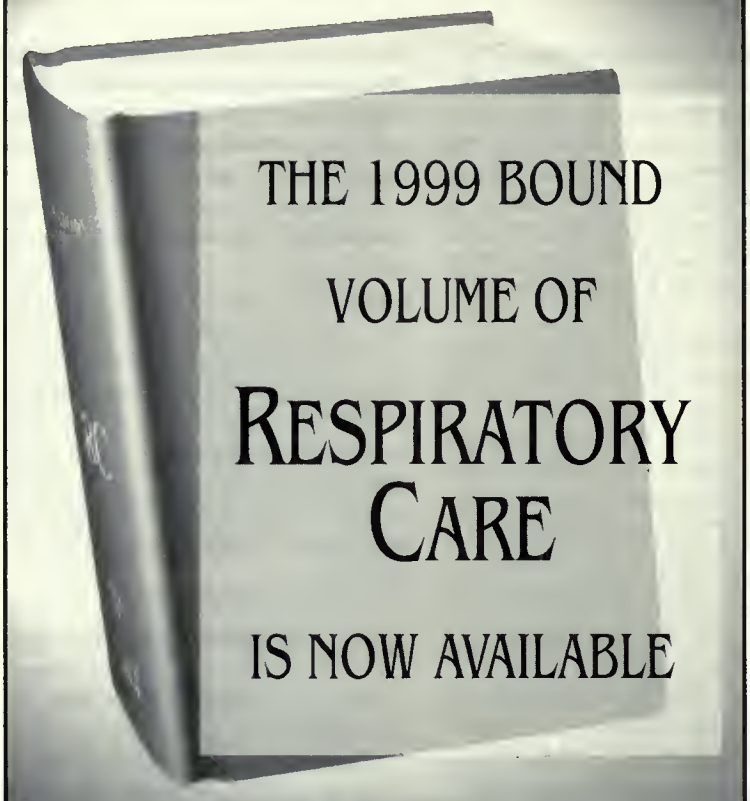

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New Products & Services

Valved Holding Chamber. Monaghan Medical Corporation announces Food and Drug Administration clearance to market its new AeroChamber Plus™ valve holding chamber product line. Monaghan describes AeroChamber Plus VHC as metered dose inhaler for asthma and COPD patients that offers new features including a "flowdynamic" system that they say increases the fine particle dose of MDIs. The new line also includes the AeroChamber Plus with mask for young patients which, the company says, provides lower inhalation resistance, less dead space, and an improved facial seal. For more information from Monaghan Medical Corporation, circle number 177 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

TB Test. Gen-Probe Incorporated has announced that the Food and Drug Administration has approved additional indication for its amplified test for *Mycobacterium tuberculosis* (TB). According to Gen-Probe, the test is now approved for the rapid diagnosis of TB in smear-negative patients. For more information from Gen-Probe Incorporated, circle number 178 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Proficiency Test Program. Accutest introduces PT STAT™, a proficiency test program. The company says that the program is designed for laboratories that measure critical care analytes and that it provides external peer group comparisons three times a year, along with a national database that includes more than 600 blood gas instruments. Accutest says tests offered include pH, blood gases, electrolytes, critical blood metabolites, hematocrit, hemoglobin, and hemoglobin fractions. For more information from Accutest, circle number 179 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

PEP System. DHD Healthcare has announced improvements to its TheraPEP® positive expiratory pressure therapy system. According to DHD, the updates make PEP more convenient for patients by providing two smaller orifice sizes and by adding the option of using an MDI or nebulizer with the device to provide medication delivery simultaneously with secretion clearance. For more information from DHD Healthcare, circle number 180 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Synchronous Digital Video. Cadwell Laboratories presents its synchronous digital video system for sleep professionals. According to the company, this new system will allow sleep professionals to more accurately correlate physical and polysomnographic events during a sleep study. Cadwell says that up to 72 hours of time-locked digital video with audio can be recorded and synchronized to the 32-channel digital PSG/EEG record at 30 frames per second. Company press materials also say that the system includes playback and editing tools that can be used to extract significant PSG/EEG data and video clips. For more information from Cadwell Laboratories, circle number 181 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

Calendar of Events

AARC & AFFILIATES

March 7—*Live Videoconference*
Participate in a live, 90-minute satellite broadcast of the first installment of the AARC's 2000 "Professor's Rounds" series and receive one CRCE credit hour (nurses earn 1.2 hours of CE credit). "Pulmonary Rehabilitation: What You Need to Know" will be broadcast from 11:30 a.m.-1 p.m. (CT). **Contact:** For more information, call the AARC at (972) 243-2272.

March 15–17—*Lake Tahoe, Nevada*
The Greater Bay Area Chapter of the California Society for Respiratory Care host their 21st annual conference at Caesars Lake Tahoe. "Tahoe 2000" will offer 12 hours of continuing education credit. **Contact:** For more information, call (925) 866-6643 or access their web site at **www.csrc.org**.

March 28—*Live Videoconference*
Participate in a live, 90-minute satellite broadcast of the first installment of the AARC's 2000 "Professor's Rounds" series and receive one CRCE credit hour (nurses earn 1.2 hours of CE credit). "Pediatric Asthma in the ER" will be broadcast from 11:30 a.m. -1 p.m. (CT). **Contact:** For more information, call the AARC at (972) 243-2272.

April 13-14—*King of Prussia, Pennsylvania*
The Pennsylvania Society for Respiratory Care presents their Third Annual Eastern Regional Conference and Exhibition at the Holiday Inn. "Diversity, Dimension, Design 2000" will feature speakers Wiliam J. Malley, MS, RRT, and AARC President Garry W. Kauffman, MPA, CHE, RRT. Ten CEUs are pending. **Contact:** Angie Herstine at (609) 784-0340, or Ann Cusano at (215) 646-7300, ext. 428.

May 9-12—*Grand Rapids, Michigan*
The Michigan Society for Respiratory Care will host their Annual Spring Conference at the Grand Center/Amway Grand Plaza Hotel.
Contact: For more information, call (517) 336-7570 or visit their web site at **www.MichiganRC.com.**

May 19—*Brainerd, Minnesota*
The Minnesota Society for Respiratory Care host their Spring Fling — "Rev It Up" at the Breezy Point Resort. Five CRCEs will be requested. **Contact:** For more information, contact Laurie Tomaszewski at (651) 232-1922, Carolyn Dunow at **dunowc@fhpcare.com**, or Carl Mottram at **mottram.carl@mayo.edu**.

May 21-22—*Spokane, Washington*
The Respiratory Care Society of Washgton will hold its 27th Annual Pacific Northwest Regional Respiratory Care Conference at Cavanaugh's Inn at the Park. Topics will include current government issues and respiratory care, high frequency, and open lung strategies.
Contact: For more information, contact Larry Knisley at (509) 921-6560, Garth Arkell at (509) 924-1197 or e-mail **arkell4@gateway.net**.

September 20-22—*Rochester, Minnesota*
The Minnesota Society for Respiratory Care host their 31st Annual Fall State Conference—"Too Hot to Handle." **Contact:** For more information, contact Laurie Tomaszewski at (651) 232-1922, Carolyn Dunow at **dunowc@fhpcare.com**, or Carl Mottram at **mottram.carl@mayo.edu**.

Other Meetings

March 27–30—*Clearwater Beach, Florida*
All Children's Hospital will host a neonatal/pediatric transport conference, "Leading the Way — Staying Patient Focused," at the Hilton Clearwater Beach Resort. Continuing education hours available for RNs and RTs.
Contact: Connie Spadaccino at (800) 456-4543, ext. 4240, or fax (727) 892-4399.

April 1-7—*Miami, Florida*
Miami Children's Hospital and the Ventilation Assisted Children's Center (VACC) are hosting their annual camp for ventilation-assisted children and their families. The campsite is A.D. Barnes Park, a 62-acre park located two miles from Miami Children's Hospital. Facilities include an air-conditioned lodge, two air-conditioned bunkhouses, and wheelchair-accessible swimming pool, playground, and nature trail. The VACC Camp treats families with children dependent on oxygen, a tracheostomy, ventilator, CPAP, or bi-level PAP to a week of fun and adventure in the company of their peers.
Contact: If you know of eligible, interested families, have them contact Camp Director Dr. Moises Simpeer or Bela Floretin at (305) 662-8380, ext. 4610, or (305) 662-8222. Applications will be accepted through Jan. 15, 2000.

May 19–21—*Atlanta, Georgia*
Children's Healthcare of Atlanta-Egleston will sponsor "Moving ECMO into the New Millennium" — SEECMO 2000, the 10th Annual Southeastern ECMO Conference, at the Grand Hyatt Buckhead. Presentations will cover adult ECMO, ECMO flow direction, CVVH techniques, and hands-on water drills.
Contact: Micheal Heard, RN, at (404) 315-2593 or **micheal.heard@choa.org**.

call for abstracts

RESPIRATORY CARE • OPEN FORUM 2000

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in Cincinnati, Ohio, October 7-10, 2000. Accepted abstracts will be published in the August 2000 issue of RESPIRATORY CARE. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report **(1) an original study, (2) the evaluation of a method, device or protocol, or (3) a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions*. Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocal valuation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. A font like Helvetica or Times makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in one paragraph on a clean sheet of paper, using margins set so that the abstract will fit into a box no bigger than 18.8 cm (7.4") by 13.9 cm (5.5"), as shown on the reverse of this page.* Insert only one letter space between sentences. Text submission on diskette is allowed but must be accompanied by a hard copy. *Data may be submitted in table form, and simple figures may be included provided they fit within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to Linda Barcus at (972) 406-4667.

Early Deadline Allowing Revision. Authors may choose to submit abstracts early. Abstracts postmarked by February 29, 2000 will be reviewed and the authors notified *by letter only* to be mailed by March 31, 2000. Rejected abstracts will be accompanied by a written critique that should, in many cases, enable authors to revise their abstracts and resubmit them by the Final Deadline (April 28, 2000).

Final Deadline. The mandatory Final Deadline is April 28, 2000 (postmark). Authors will be notified of acceptance or rejection *by letter only*. These letters will be mailed by July 12, 2000.

Mailing Instructions. Mail (Do not fax!) 2 clear copies of the completed abstract form, diskette (if possible), and a stamped, self-addressed postcard (for notice of receipt) to:

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American Association for Respiratory Care

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Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days.

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An individual is eligible if he/she lives in the U.S. or its territaries ar was an Active Member priar ta moving outside its borders or territories, and meets ONE af the following criteria: (1) is legally credentialed as a respiratory core prafessianal if emplayed in o state that mandates such, OR (2) is a graduate af an accredited educatianal program in respiratory care, OR (3) halds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

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- ☐ 1 year
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- ☐ 4 years
- ☐ Other, specify _____

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Month _____ Year _____

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We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
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- ☐ RPFT
- ☐ Perinotal/Pediatric

Salary

- ☐ Less than $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

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NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

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Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

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Research Article: A report of an original investigation (a study). Must include Title Page, Abstract, Key Words, Background, Methods, Results, Discussion, Conclusions, and References. May also include Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendices.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a topic that has been the subject of at least 40 published research articles. Must include: Title Page, Outline, Key Words, Introduction, Review of the Literature, Summary, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

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Update: A report of subsequent developments in a topic that has been critically reviewed in RESPIRATORY CARE or elsewhere. Same structure as a Review Article.

Special Article: A pertinent paper not fitting one of the other categories. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper addressing an issue in the practice or administration of respiratory care. It may present an opposing opinion, clarify a position, or bring a problem into focus.

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> Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

> Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

> American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)

> Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986; 89(3 Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided, and those more than 3 years old should not be cited.)

> Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in a journal:

> Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

> Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

> Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Corporate author book:

> American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

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> DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990:76-85.

Chapter in book with editor(s):

> Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott; 1977:26-42.

Paper accepted but not yet published:

> Hess D. New therapies for asthma. Respir Care (year, in press).

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FDA Use Only (Reap Care)

Triage unit sequence #

A. Patient information

1. Patient identifier
In confidence

2. Age at time of event:
or
Date of birth:

3. Sex
☐ female
☐ male

4. Weight
____ lbs
or
____ kgs

B. Adverse event or product problem

1. ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)
☐ death ____ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other: ____

3. Date of event (mo/day/yr)

4. Date of this report (mo/day/yr)

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
☐ health professional
☐ lay user/patient
☐ other: ____

5. Expiration date (mo/day/yr)

6.
model # ____
catalog # ____
serial # ____
lot # ____
other #

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9. Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ____ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name & address

phone #

2. Health professional?
☐ yes ☐ no

3. Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

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DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
Hubert H. Humphrey Building, Room 531-H
200 Independence Avenue, S.W.
Washington, DC 20201

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Please do NOT return this form to either of these addresses.

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Public Health Service • Food and Drug Administration

FDA Form 3500-back

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Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

Scheduled Professor's Rounds 2000

Pulmonary Rehabilitation: What You Need to Know— Julien M Roy BA RRT; Host, Richard Branson BA RRT—Video March 7; Audio April 4

Pediatric Asthma in the ER—Tim Myers BS RRT; Host, Richard Branson BA RRT—Video March 28; Audio April 18

Drugs, Medications and Delivery Devices of Importance in Respiratory Care—Jim Fink MS RRT; Host, David Pierson MD—Video April 25; Audio May 16

Cost Effective Respiratory Care: You've Got to Change—Kevin Shrake MA RRT FACHE; Host, Sam P Giordano MBA RRT—Video May 23;Audio June 20

Pediatric Ventilation: Kids Are Different—Mark Heulitt MD; Host, Richard Branson BA RRT—Video July 25; Audio August 15

What Matters in Respiratory Monitoring: What Goes and What Stays—Dean Hess PhD RRT FAARC; Host, Richard Branson BA RRT—Video August 22; Audio September 26

Managing Asthma: An Update—Patti Joyner RRT CCM; Host, Mari Jones MSN RN RRT—Video September 19; Audio October 17

Routine Pulmonary Function Testing: Doing it Right—Carl D Mottram RRT RPFT; Host, David Pierson MD—Video November 7; Audio December 5

Helpful Websites

American Association for Respiratory Care
http://www.aarc.org
- Current job listings
- American Respiratory Care Foundation fellowships, grants, & awards
- Clinical Practice Guidelines

National Board for Respiratory Care
http://www.nbrc.org

RESPIRATORY CARE online
http://www.rcjournal.com
- Subject and Author Indexes
- Contact the editorial staff

Asthma Management Model System
http://www.nhlbi.nih.gov

Keys to Professional Excellence
http://www.aarc.org/keys/

The National Board for Respiratory Care—2000 Examination Dates and Fees

Examination	Applications Accepted/ Testing Available	Examination Fees
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Perinatal/Pediatric	Dec 1, 1999 Jan 10, 2000	$250 (new applicant) $220 (reapplicant)
CPFT	Jan 1, 2000 Feb 1, 2000	$200 (new applicant) $170 (reapplicant)
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RRT (Written & CSE)	Feb 1, 2000 Apr 1, 2000	$190 (new - written only) $200 (new - CSE only) $390 (new - both)

For information about other services or fees, write to the National Board for Respiratory Care, 8310 Nieman Road, Lenexa KS 66214, or call (913) 599-4200, FAX (913) 541-0156,or e-mail: nbrc-info@nbrc.org

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Pulmonetic Systems, Inc.	Ventilator	128	800-754-1914	C3
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